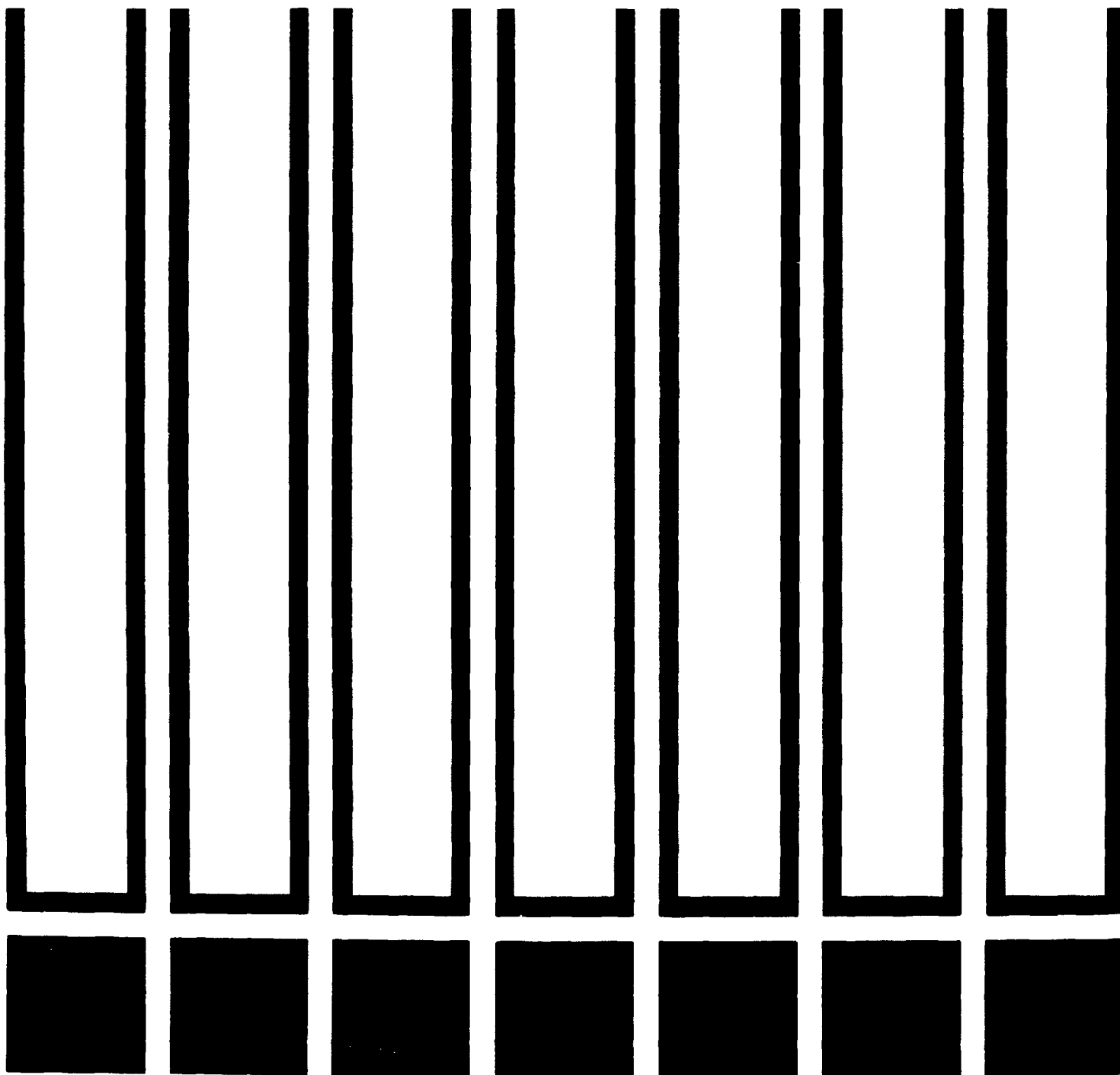


NIOSH

criteria for a recommended standard
occupational exposure to
ASPHALT FUMES



U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

Public Health Service Center for Disease Control

National Institute for Occupational Safety and Health

criteria for a recommended standard....

OCCUPATIONAL EXPOSURE TO ASPHALT FUMES



U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

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National Institute for Occupational Safety and Health

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PREFACE

The Occupational Safety and Health Act of 1970 emphasizes the need for standards to protect the health and safety of workers exposed to an ever-increasing number of potential hazards at their workplace. The National Institute for Occupational Safety and Health has projected a formal system of research, with priorities determined on the basis of specified indices, to provide relevant data from which valid criteria for effective standards can be derived. Recommended standards for occupational exposure, which are the result of this work, are based on the health effects of exposure. The Secretary of Labor will weigh these recommendations along with other considerations such as feasibility and means of implementation in developing regulatory standards.

It is intended to present successive reports as research and epidemiologic studies are completed and as sampling and analytical methods are developed. Criteria and standards will be reviewed periodically to ensure continuing protection of the worker.

I am pleased to acknowledge the contributions to this report on asphalt fumes by members of the NIOSH staff and the valuable constructive comments by the Review Consultants on Asphalt Fumes, by the ad hoc committees of the Society of Toxicology and the American Academy of Industrial Hygiene, and by Robert B. O'Connor, M.D., NIOSH consultant in

occupational medicine. The NIOSH recommendations for standards are not necessarily a consensus of all the consultants and professional societies that reviewed this criteria document on asphalt fumes. A list of Review Consultants appears on page vi.



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The Division of Criteria Documentation and Standards Development, National Institute for Occupational Safety and Health, had primary responsibility for the development of the criteria and recommended standard for asphalt fumes. Craig R. McCormack of this Division served as criteria manager. SRI International developed the basic information for consideration by NIOSH staff and consultants under contract CDC 99-74-31.

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The views expressed and conclusions reached in this document, together with the recommendations for a standard, are those of NIOSH. These views and conclusions are not necessarily those of the consultants, other federal agencies and professional societies that reviewed the document, or of the contractor.

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CRITERIA DOCUMENT:
RECOMMENDATIONS FOR AN OCCUPATIONAL
EXPOSURE STANDARD FOR ASPHALT FUMES

Table of Contents

	<u>Page</u>
PREFACE	111
REVIEW CONSULTANTS	vi
I. RECOMMENDATIONS FOR AN ASPHALT FUMES STANDARD	1
Section 1 - Environmental (Workplace Air)	4
Section 2 - Medical	4
Section 3 - Labeling and Posting	5
Section 4 - Personal Protective Equipment and Clothing	7
Section 5 - Informing Employees of Hazards from Asphalt Fumes	10
Section 6 - Work Practices	10
Section 7 - Sanitation Practices	13
Section 8 - Monitoring and Recordkeeping	13
II. INTRODUCTION	16
III. BIOLOGIC EFFECTS OF EXPOSURE	19
Extent of Exposure	22
Historical Reports	23
Effects on Humans	25
Epidemiologic Studies	31
Animal Toxicity	35
Correlation of Exposure and Effect	68
Carcinogenicity, Mutagenicity, Teratogenicity, and Effects on Reproduction	71
Summary Tables of Exposure and Effect	74
IV. ENVIRONMENTAL DATA	75
Environmental Concentrations	75
Sampling and Analysis	75
Engineering Controls	80
V. WORK PRACTICES	82

Table of Contents (Continued)

	<u>Page</u>
VI. DEVELOPMENT OF STANDARD	87
Basis for Previous Standards	87
Basis for the Recommended Standard	89
VII. RESEARCH NEEDS	99
VIII. REFERENCES	102
IX. APPENDIX I - Method for Sampling Asphalt Fumes in Air	109
X. APPENDIX II - Analytical Method (Total Particulate) for Asphalt Fumes	113
XI. APPENDIX III - Analytical Method (Solvent Extraction) for Asphalt Fumes	116
XII. APPENDIX IV - Material Safety Data Sheet	121
XIII. TABLES AND FIGURES	131

I. RECOMMENDATIONS FOR AN ASPHALT FUMES STANDARD

The National Institute for Occupational Safety and Health (NIOSH) recommends that employee exposure to asphalt fumes in the workplace be controlled by adherence to the following sections. The standard is designed to protect the health and provide for the safety of employees for up to a 10-hour work shift, 40-hour workweek, over a working lifetime. Compliance with all sections of the standard should prevent adverse effects of asphalt fumes on the health of employees and provide for their safety. The standard is measurable by techniques that are valid, reproducible, and available to industry and governmental agencies. Sufficient technology exists to permit compliance with the recommended standard. Although the workplace occupational exposure limit is considered to be a safe level based on current information, it should be regarded as the upper boundary of exposure and every effort should be made to maintain exposure at a level as low as is technically feasible. The criteria and standard will be subject to review and revision as necessary.

The principal adverse effects on health from exposure to asphalt fumes are irritation of the serous membranes of the conjunctivae and the mucous membranes of the respiratory tract. Hot asphalt can cause burns of the skin. In animals, there is evidence that asphalt left on the skin for long periods of time may result in local carcinomas, but there have been no reports of such effects on human skin that can be attributed to asphalt alone. No reliable reports of malignant tumors of parenchymatous organs due to exposure to asphalt fumes have been found, but there has been no

extensive study of this possible consequence of occupational exposure in the asphalt industry.

Asphalt fumes are defined as the cloud of small particles created by condensation from the gaseous state after volatilization of asphalt. Approximately 96% of the asphalt used in this country is used in paving and roofing operations. "Occupational exposure" to asphalt fumes is defined as exposure in the workplace at a concentration of one-half or more of the recommended occupational exposure limit. If exposure to other chemicals also occurs, as is the case when asphalt is mixed with a solvent, emulsified, or used concurrently with other materials such as tar or pitch, provisions of any applicable standard for the other chemicals shall also be followed.

A gravimetric method is recommended for estimation of the air concentration of asphalt fumes. When large amounts of dust are present in the same atmosphere in which the asphalt fume is present, which may occur in road-building operations, the gravimetric method may lead to erroneously high estimates for asphalt fumes, and to possibly undeserved sanctions and citations for ostensibly exceeding the environmental limit for asphalt fumes or nuisance particulates. NIOSH recommends that where the resolution of such problems becomes necessary, a more specific procedure, which involves solvent extraction and gravimetric analysis, be employed for the determination of asphalt fumes. The best procedure now available seems to be ultrasonic agitation of the filter in benzene and weighing of the dried residue from an aliquot of the clear benzene extract. NIOSH is attempting to devise an even more specific method for asphalt fumes for use under such conditions.

It is possible, but not proven, that benzene can be replaced without loss of extraction efficiency by other solvents, including cyclohexane.

IF BENZENE IS USED, THE FACT THAT NIOSH REGARDS THIS CHEMICAL AS A VIRTUAL CARCINOGEN SHOULD BE KEPT IN MIND. PRECAUTIONS SHOULD BE TAKEN TO PREVENT EXPOSURE OF ANALYTIC AND OTHER PERSONNEL TO SIGNIFICANT AMOUNTS OF BENZENE AS EITHER LIQUID OR VAPOR. Because existing controls in laboratories, eg, fume hoods, are likely to be insufficient to control benzene to the extent necessary, it is proposed that cyclohexane be used as the solvent for extracting constituents of asphalt in the analytical procedure. Whether cyclohexane will extract these constituents efficiently, ie, whether cyclohexane extraction procedures are as efficient as benzene extraction procedures in monitoring exposure to asphalt fumes, needs to be determined experimentally. If the procedures are found to be inefficient, a solvent other than cyclohexane, conceivably even benzene, may be recommended.

When mineral dusts are present in comparatively high amounts on the sampling filters, the possible contributions of silica dust and asbestos microfibers to pulmonary disease should be remembered and appropriate steps taken to minimize the exposure of employees to such dusts.

The small number of papers reporting a biologic hazard from exposure to asphalt fumes has not allowed a determination of the concentration threshold that produces adverse effects. Because of the presence of minute quantities of neoplastigenic hydrocarbons in some asphalts, NIOSH believes that asphalt fumes should be considered to be somewhat more hazardous than a nuisance dust. Accordingly, a ceiling concentration limit numerically

equivalent to the current US occupational exposure limit for respirable nuisance particulate is recommended.

Section 1 - Environmental (Workplace Air)

(a) Concentration

Occupational exposure to asphalt fumes shall be controlled so that employees are not exposed to the airborne particulates at a concentration greater than 5 milligrams per cubic meter of air (mg/cu m), determined during any 15-minute period.

(b) Sampling and Analysis

Occupational environmental samples shall be collected and analyzed as described in Appendices I and II or III, or by any method shown to be equivalent in accuracy, precision, and sensitivity to the methods specified.

Section 2 - Medical

Medical surveillance shall be made available as outlined below to all workers subject to occupational exposure to asphalt fumes.

(a) Preplacement examinations shall include at least:

(1) Comprehensive medical and work histories with special emphasis directed towards the eye, skin, and respiratory system.

(2) Physical examination giving particular attention to evidence of abnormalities in the eyes, skin, or respiratory system.

(3) A judgment of the worker's ability to use positive and negative pressure respirators.

(b) Periodic examinations shall be made available at a frequency to be determined by the responsible physician. These examinations shall include at least:

(1) Interim medical and work histories.

(2) Physical examination as outlined in (a)(2) above.

(c) During examinations, applicants or employees found to have medical conditions which would be directly or indirectly aggravated by exposure to asphalt fumes shall be counseled on the increased risk of impairment of their health by working with this substance.

(d) Initial medical examinations shall be made available to all workers as soon as practicable after the promulgation of a standard based on these recommendations.

(e) In the event of illness known or suspected to be due to asphalt fumes, a physical examination shall be made available.

(f) Pertinent medical records shall be maintained for all employees exposed to asphalt fumes in the workplace. Such records shall be kept for at least 30 years after termination of employment. These records shall be made available to the designated medical representatives of the Secretary of Health, Education, and Welfare, of the Secretary of Labor, of the employer, and of the employee or former employee.

Section 3 - Labeling and Posting

All labels and warning signs shall be printed both in English and in the predominant language of non-English-reading workers. Illiterate workers and workers reading languages other than those used on labels and posted signs shall receive information regarding hazardous areas and shall be informed of the instructions printed on the labels and signs.

(a) Labeling

The following labels shall be affixed in a readily visible position on all tanks and containers of hot asphalt:

HOT ASPHALT

WARNING!
MAY CAUSE SEVERE BURNS

Do not get in eyes or on skin.
Use only with adequate ventilation.
Wear safety glasses, face shield, gloves, and protective clothing when handling.

First Aid: Call a physician as quickly as possible. In case of contact with eyes, immediately flush eyes with plenty of water for at least 15 minutes. In case of contact with skin, immerse affected part in cold water. If ice is available, pack ice on the asphalt adhering to the skin or apply an emergency cold pack. If difficulty in breathing occurs after inhalation, remove victim to fresh air and keep warm and quiet. If breathing stops, give artificial respiration.

The following labels shall be affixed in a readily visible position on all tanks and containers of cold liquid asphalt:

(*ASPHALT)

WARNING!
HARMFUL IF SWALLOWED
MAY BE IRRITATING TO SKIN AND EYES

Do not get in eyes or on skin.
Do not take internally.
Use only with adequate ventilation.
Wear safety glasses, face shield, gloves, and protective clothing when handling.

First Aid: Call a physician as quickly as possible. In case of contact with eyes, flush eyes immediately with plenty of water for at least 15 minutes. In case of contact with skin, wash affected area with detergent and water. If swallowed, give milk or olive oil. DO NOT INDUCE VOMITING.

*Insert CUT-BACK or EMULSIFIED, whichever is appropriate

(b) Posting

(1) The following warning sign shall be posted in readily visible locations at or near all entrances to areas where asphalt fumes are generated, and on or near all equipment generating asphalt fumes:

WARNING!
ASPHALT FUMES EXPOSURE AREA

HIGH CONCENTRATIONS OF FUMES MAY
CAUSE NOSE AND EYE IRRITATION

FIRE HAZARD

(2) If respirators are required for protection from asphalt fumes, the following statement shall be added in large letters to the sign required in Section 3(b):

RESPIRATORY PROTECTION REQUIRED IN THIS AREA

(3) In any workroom or area where it is likely that emergency situations will arise from accidental skin or eye contact or other excessive exposure to asphalt or asphalt fumes, signs required by Section 3(b) shall be supplemented by additional signs giving emergency and first-aid instructions and procedures and the location of first-aid supplies and emergency equipment, including respiratory protective equipment.

Section 4 - Personal Protective Equipment and Clothing

(a) Respiratory Protection

(1) Engineering controls shall be used when needed to keep concentrations of asphalt fumes below the recommended exposure limit. The

only conditions under which compliance with the recommended exposure limit may be achieved by the use of respirators are:

(A) During the time required to install or test the necessary engineering controls.

(B) For operations such as nonroutine maintenance or repair activities causing brief exposure at concentrations above the environmental limit.

(C) During emergencies when concentrations of asphalt fumes may exceed the environmental limit.

(2) When a respirator is permitted by paragraph (a)(1) of this section, it shall be selected from a list of respirators approved under 30 CFR 11 and used in accordance with the following requirements:

(A) The employer shall establish and enforce a respiratory protective program meeting the requirements of 29 CFR 1910.134.

(B) When employees are exposed above the recommended limit, the employer shall provide respirators in accordance with Table I-1 and shall ensure that the employees use the respirators provided.

(C) Respirators specified for use in higher concentrations of asphalt fumes may be used in atmospheres of lower concentrations.

(D) The employer shall ensure that respirators are adequately cleaned and maintained and that employees are instructed and drilled at least annually in the proper use and testing for leakage of respirators assigned to them.

TABLE I-1

RESPIRATOR SELECTION GUIDE FOR ASPHALT FUMES

Concentration (mg/cu m)	Respirator Type Approved under Provisions of 30 CFR 11
Less than or equal to 25	(1) Single-use dust respirator or quarter- mask dust respirator (2) Supplied-air respirator operated in de- mand mode (negative pressure) equipped with quarter-mask facepiece
Less than or equal to 50	(1) Half-mask facepiece dust respirator (2) Supplied-air respirator operated in de- mand mode (negative pressure) equipped with half-mask facepiece, or supplied-air respi- rator operated in continuous-flow mode equipped with half-mask facepiece
Greater than 50 or for emergency entry such as firefighting	Self-contained breathing apparatus operated in pressure-demand or other positive pres- sure mode

(E) Respirators shall be easily accessible, and employees shall be informed of their location.

(F) When a self-contained breathing apparatus is used in atmospheres with asphalt fumes concentrations greater than 50 mg/cu m, standby workers with suitable rescue and communications equipment must be present as provided in Section 6(b)(4).

(b) Protective Clothing

Employees shall wear appropriate protective clothing, including gloves, suits, boots, face shields (8-inch minimum), or other clothing as needed, to prevent eye and skin contact with asphalt.

Section 5.- Informing Employees of Hazards from Molten Asphalt
and Asphalt Fumes

(a) The employer shall provide to the employee at the beginning of employment and on a periodic basis thereafter information concerning hazards, relevant symptoms of overexposure, appropriate emergency procedures, and proper conditions and precautions necessary to minimize exposure to molten asphalt and asphalt fumes. Employees shall be provided with up-to-date information whenever there is a process change. Records of such training shall be kept to verify the frequency of training, and each employee shall be advised of the availability of such information, which shall include that prescribed in paragraph (b) of this section and shall be accessible to the worker at each establishment or department where asphalt fumes may be generated.

(b) Required information shall be recorded as specified in Appendix IV, "Material Safety Data Sheet," or on a similar form approved by the Occupational Safety and Health Administration, US Department of Labor.

Section 6 - Work Practices

(a) Emergency Procedures

For all work areas in which there is a potential for emergencies involving asphalt fumes, the procedures specified below, as well as any other procedures appropriate for a specific operation or process, shall be formulated in advance, and employees shall be instructed in their implementation.

(1) Procedures shall include prearranged plans for obtaining emergency medical care and for necessary transportation of

injured workers. Employees shall also be trained in administering immediate first aid and shall be prepared to render such assistance when necessary.

(2) Personnel essential to emergency operations shall use approved skin and respiratory protection as specified in Section 4.

(3) Employees not essential to emergency operations shall be evacuated from exposure areas during emergencies. Perimeters of hazardous exposure areas shall be delineated, posted, and secured.

(4) All persons who may be required to shut off sources of asphalt fumes and to repair leaks shall be properly trained in emergency procedures and adequately protected against the attendant hazards from exposure to asphalt fumes.

(b) Confined Spaces

(1) Entry into confined spaces, such as tanks, process vessels, and tunnels, shall be controlled by a permit system. Permits signed by an authorized representative of the employer shall certify that preparation of the confined space, precautionary measures, and personal protective equipment are adequate and that precautions have been taken to ensure that prescribed procedures will be followed.

(2) Confined spaces that have previously contained asphalt fumes shall be inspected and tested for the presence of asphalt fumes and the temperature shall be measured prior to entry.

(3) Confined spaces shall be ventilated while work is in progress to keep the concentrations of asphalt fumes and other air contaminants below the workplace occupational exposure limits and to assure an adequate supply of oxygen. Air from the confined spaces shall be

ventilated to a point remote from any work area. When ventilation is inadequate to maintain the concentration of asphalt fumes and other air contaminants below the recommended occupational exposure limits, respiratory protective equipment shall be used in accordance with the provisions of Table I-1.

(4) Any individual entering confined spaces where the concentration of asphalt fumes may exceed 50 mg/cu m or where other air contaminants are excessive shall wear a suitable harness with lifelines tended outside the space by another employee who shall also be equipped with the necessary protective equipment, including a self-contained breathing apparatus that operates in the pressure-demand (positive pressure) mode. Communication (visual, voice, signal line, telephone, radio, or other suitable means) with the employee inside the enclosed space shall be maintained by the standby person.

(c) Engineering Controls

Engineering controls, such as local exhaust ventilation, shall be used to keep concentrations of asphalt fumes and other air contaminants below the recommended occupational exposure limits and to provide oxygen. Powered ventilation systems shall be designed to prevent the accumulation of asphalt fumes in the workplace. Ventilation systems shall be subject to regular preventive maintenance and cleaning to ensure maximum effectiveness (which shall be verified by periodic airflow measurements) and prevent the occurrence of fires in the accumulated asphalt. The system efficiency measurements shall also be made within 5 workdays of any change in production or control equipment that might result in an increase in the concentrations of asphalt fumes. Before maintenance work on control

equipment begins, sources of asphalt fumes shall be eliminated to the extent feasible. If concentrations below the recommended occupational exposure limits cannot be assured, respiratory protective equipment as specified in Table I-1 shall be used during nonroutine maintenance work. The employer shall ensure that the required measurements are performed by technically qualified persons.

Section 7 - Sanitation Practices

(a) Eating and food preparation or dispensing (including vending machines) shall be prohibited in the immediate area of asphalt use or where asphalt fumes are present.

(b) Smoking shall be prohibited in the immediate area of asphalt use or where asphalt fumes are present.

(c) Employees who handle asphalt or who work in an area where they are exposed to asphalt fumes shall be instructed to wash their hands with nonvolatile skin cleaners and water before drinking, eating, smoking, or using toilet facilities.

Section 8 - Monitoring and Recordkeeping

Workers shall not be considered occupationally exposed to asphalt fumes if environmental concentrations, as determined on the basis of an industrial hygiene survey conducted as soon as practicable after the promulgation of a standard based on these recommendations, do not exceed one-half the recommended occupational exposure limit. Records of these surveys, including the basis for concluding that air levels are at or below

this limit, shall be kept. Surveys shall be repeated at least once every 3 years and within 30 days after any process change likely to result in an increased concentration of asphalt fumes.

If it has been determined that environmental concentrations of asphalt fumes may exceed one-half the recommended occupational exposure limit, the following requirements apply:

(a) Personal Monitoring

(1) A program of personal monitoring shall be instituted to identify and measure, or permit calculation of, the exposure of all employees occupationally exposed to asphalt fumes.

(2) Routine monitoring of occupationally exposed employees shall be conducted at least annually.

(3) If monitoring of an employee's exposure to asphalt fumes reveals an exposure in excess of the recommended ceiling concentration limit, control measures shall be initiated, the exposure of that employee shall be measured at least every 30 days, the employer shall ensure that the employee is protected by a respirator, and the employee shall be notified of the exposure and of the control measures being implemented. Such monitoring shall continue until two consecutive determinations, at least a week apart, indicate that the employee's exposure no longer exceeds the recommended environmental limit. Routine monitoring may then be resumed.

(4) In all personal monitoring, samples representative of the exposure in the breathing zone of the employee shall be collected. Procedures for sampling, calibration of equipment, and analysis of samples of air for asphalt fumes shall be in accordance with Section 1(b).

(5) For each determination of an occupational exposure concentration, a sufficient number of samples shall be taken to characterize the employee's exposure during each work shift. Variations in work and production schedules and in the employee's location and job functions shall be considered when samples are collected.

(b) Recordkeeping

Employers or their successors shall keep records of environmental monitoring for each employee for at least 30 years after the individual's employment has ended. These records shall include the name and social security number of the employee being monitored, duties and job locations within the worksite, dates of measurements, sampling and analytical methods used and evidence for their accuracy, duration of sampling, number of samples taken, results of analyses, occupational exposure concentrations based on these samples, and any personal protective equipment used by the employee. Records for each employee, indicating date of employment with the company and changes in job assignment, shall be kept for the same 30-year period. The employer shall make these records available upon request to authorized representatives of the Assistant Secretary of Labor for Occupational Safety and Health or of the Director of the National Institute for Occupational Safety and Health. Employees or their authorized representatives shall have access to information on their own exposures and shall be given the opportunity to observe any measurement conducted in accordance with this section.

II. INTRODUCTION

This report presents the criteria and the recommended standard based thereon that were prepared to meet the need for preventing occupational disease or injury arising from exposure to asphalt fumes. The criteria document fulfills the responsibility of the Secretary of Health, Education, and Welfare under Section 20(a)(3) of the Occupational Safety and Health Act of 1970 to "...develop criteria dealing with toxic materials and harmful physical agents and substances which will describe...exposure levels at which no employee will suffer impaired health or functional capacities or diminished life expectancy as a result of his work experience."

The National Institute for Occupational Safety and Health (NIOSH), after a review of data and consultation with others, formalized a system for the development of criteria upon which standards can be established to protect the health and to provide for the safety of employees exposed to hazardous chemical and physical agents. The criteria and the recommended standard should enable management and labor to develop better engineering controls resulting in more healthful work practices and should not be used as a final goal.

Irritation of the serous membranes of the conjunctivae and the mucous membranes of the respiratory tract are the principal adverse effects on health from exposures to asphalt fumes. Hot asphalt can cause burns of the skin. There is evidence from animal experiments that local carcinomas may result from asphalt when it is left on the skin for long periods of time. However, there have been no reports of such effects on human skin that can

be attributed to exposures to asphalt alone. Reliable reports associating malignant tumors of parenchymatous organs with exposure to asphalt fumes have not been found in the literature. Although available information has not clearly demonstrated that a direct carcinogenic hazard is associated with asphalt fumes, NIOSH is concerned that future investigations may suggest a greater occupational hazard from asphalt fumes than is currently documented in the literature. The lack of credible toxicologic evidence and the confusion in the literature regarding asphalts, tars, and pitches have precipitated this concern. The toxic effects produced by the three substances are quantitatively and qualitatively different, and in order to afford workers adequate protection, special care must be exercised in determining and defining the exposure material. In view of the unresolved issues in this particular area, NIOSH is compelled to recommend a ceiling concentration limit of 5 mg/cu m, based on total particulate, for asphalt fumes.

These criteria for a recommended standard for asphalt fumes are developed as part of a continuing series of documents published by NIOSH. The recommended standard applies to workplace exposure to asphalt fumes arising from the handling, processing, manufacture, use, or storage of asphalt. The standard was not designed for the population-at-large, and any application to situations other than occupational exposures is not warranted. It is intended to (1) protect workers exposed to asphalt fumes against irritation of the eyes, skin, and respiratory tract, (2) be measurable by techniques that are valid, reproducible, and available to industry and government agencies, and (3) be attainable with existing technology.

The major concern in occupational exposure to asphalt fumes is their potential for irritating the eyes and respiratory tract. These effects have only been reported after exposures at unknown, high concentrations, and the available literature does not permit the determination of the thresholds for adverse effects.

Retrospective and prospective epidemiologic studies are needed to assess the potential occupational hazard from asphalt fumes. These studies must consider the possible influence of individual sensitivity, predisposing factors, such as excessive alcohol use, smoking, and obesity, and previous or concurrent exposure of employees to other, more toxic materials, such as tar and pitch. Animal experiments that simulate the exposure schedules of a workplace environment should be conducted to determine maximum safe exposures. These experiments might then be useful in estimating exposure limits for humans. Further studies are also needed to investigate the carcinogenic, mutagenic, and teratogenic potentials of asphalt fumes, to improve the recommended sampling and analytical methods, and to develop, if possible, a method for biologic monitoring of significant exposure to asphalt fumes.

III. BIOLOGIC EFFECTS OF EXPOSURE

Fumes have been defined by Patty [1] as "solid particles generated by condensation from the gaseous state, generally after volatilization from melted substances and often accompanied by a chemical reaction, such as oxidation." The American Society for Testing and Materials [2] defined asphalt as "a dark brown to black cementitious material in which the predominating constituents are bitumens which occur in nature or are obtained in petroleum processing." Asphalt fumes then, for the purposes of this document, are defined as the nimbose effusion of small, solid particles created by condensation from the vapor state after volatilization of asphalt. In addition to particles, a cloud of fume may contain materials still in the vapor state.

The chemical composition of asphalts derived from petroleum varies, depending on the source of the crude oil used to manufacture them [3-7] and the refining process and physical specifications of the finished product [5,8-10]. The major constituent groups of asphalt are asphaltenes, resins, and oils made up of saturated and unsaturated hydrocarbons [7,11]. These major constituents are soluble in carbon tetrachloride and benzene [6], but are not completely soluble in saturated hydrocarbons such as naphtha, hexane, pentane, propane, and n-heptane [4]. The asphaltenes have molecular weights in the range 1,000-2,600, those of the resins fall in the range 370-500, and those of the oils in the range 290-630. Natural asphalts contain considerable amounts of geologic material (rocks, sand, etc) and comparatively smaller amounts of bituminous substances (4-40%); the bitumens of lake asphalts have molecular weights in the range 620-1132.

Small quantities of inorganic materials may also be present, some possibly as organometallic compounds [5,7,9]. Fumes from some representative asphalts have been analyzed, and their chemical compositions are given in Table XIII-1 [5]. The amounts of benzo(a)pyrene found in fumes collected from two different plants that prepared hot mix asphalt ranged from 3 to 22 nanograms/cu m; this is approximately 0.03% of the amount in coke oven emissions and 0.01% of that emitted from coal-burning home furnaces.

Petroleum asphalt is the residue in the fractional distillation of crude oil [9,12,13]. Crude oil is distilled to fractionate the parent mixture into economically valuable distillates, such as naphtha, gasoline, kerosene, and fuel oil. Asphalt is also derived from crude oil by solvent precipitation and by air-blowing. The solvent precipitation process utilizes propane or propane-butane mixtures under controlled conditions to precipitate the asphalt from a residuum stock. The process consists of a countercurrent liquid-liquid extraction using solvent-to-oil ratios of from 4:1 to 10:1. Temperatures in this process are selected to allow suitable viscosity of the propane-oil mixture for handling in the deasphalting tower. The primary objective in this production process is the recovery of either lube or catalytic-cracking feedstock oils, rather than the production of asphalts. Air-blowing is a process for further refining an asphalt stock, termed a flux. Air at temperatures of 400-550 F (204-288 C) is bubbled through the flux, producing dehydrogenation and polymerization reactions. The air-blowing process is generally used to manufacture asphaltic paving binders and saturants for the flooring, roofing, and insulation industries from soft flux stocks. The process allows a consumer to make various saturant grades from the same base flux. Asphalts can also

be further refined, blended with the lighter petroleum fractions, or emulsified with water, depending on the use for which they are designed. Liquid asphalts are produced by blending a diluent with the asphalt. The common diluents are solvents with boiling points close to those of "gas oil," kerosene, or naphtha; asphalts formulated in this manner are termed "cut-back" asphalts. Asphalt in liquid form is also supplied as an emulsion with water. A petroleum asphalt refinery flow chart is shown in Figure XIII-1 [8].

Asphalt has often been confused with tar because the two are similar in appearance and have been used interchangeably as construction materials. Tars, however, are produced by destructive distillation of such organic materials as coal, oil, lignite, peat, or wood [2], whereas petroleum asphalt is produced as the residue from fractional distillation of crude oil. There is obvious confusion in the literature between asphalt and coal tar, and the classification of the two basic substances is often unclear [14-17]. However, even limited chemical analyses show the two substances to be quite different, especially in their proportions of polynuclear aromatic hydrocarbons and known carcinogenic chemicals [3,18]. Asphalt has also been confused with pitch, which is the residue from fractional distillation of tar [2].

The existing confusion in the use of the terms asphalt, tar, and pitch has necessitated a careful review of the literature to determine the nature of the compound that was then under discussion. In many cases, such review has shown that substances listed by the authors as asphalt have really been tars or pitches by the present definition [2], so that these papers are not relevant to this document. In cases where authors have not

provided information about the source of the compound tested, their papers are included in this document on the basis of the authors' terminology and this fact is pointed out in the review. Several papers have presented data from experiments in which animals were exposed to either asphalt or coal tar [3,17,19], or in which the adverse effects in humans from exposure to these two materials were discussed [16,18,20]. The data concerning coal tar exposure in these reports have been included in this chapter, because of the confusion in the terminology and to allow a comparison of the toxicities of coal tar and asphalt.

Extent of Exposure

Occupational exposure to asphalt fumes can occur during the transport, storage, production, handling, or use of asphalt. The composition of the asphalt that is produced is dependent on the refining process applied to the crude oil, the source of the crude oil, and the penetration grade (viscosity) and other physical characteristics of the asphalt required by the consumer [9].

The process for production of asphalt is essentially a closed-system distillation [9]. Refinery workers are therefore potentially exposed to the fumes during loading of the asphalt for transport from the refinery, during routine maintenance, such as cleaning of the asphalt storage tanks, or during accidental spills. Most asphalt is used out of doors, in paving and roofing, and the workers' exposure to the fumes is dependent on environmental conditions, work practices, and other factors. These exposures are stated to be generally intermittent and at low concentrations [21]. Workers are potentially exposed also to skin and eye contacts with

hot, cut-back, or emulsified asphalts. Spray application of cut-back or emulsified asphalts may involve respiratory exposure also.

Asphalt sales in the United States have increased yearly from approximately 3 million tons in 1926 [8] to over 27 million tons in 1975 [22]. Of the asphalt produced in 1975, 77.9% was used for paving, 17.4% for roofing, and 4.7% for miscellaneous purposes, such as insulating and waterproofing [22]. Some uses and applications of asphalt are given in Table XIII-2 [8]. Because of the nature of the major uses of asphalt and asphalt products, it is not possible to determine accurately the number of workers potentially exposed to asphalt fumes in the United States. NIOSH has not estimated the number of workers exposed to asphalt fumes, but an estimate of 500,000 can be derived from estimates of the number of workers in various occupations presented in County Business Patterns, 1973 [23].

Historical Reports

Natural asphalt was used prior to 540 BC as a cement for masonry and street construction and as a waterproofing layer for water tanks [8]. Natural asphalt was first used in the United States in sidewalk construction around 1838 and in pavement construction around 1870. The discovery in 1902 that California crude oil yielded an appreciable quantity of semisolid and solid distillation residues increased the use of petroleum asphalt so that, by 1907, the tonnage of petroleum-derived asphalt equaled that from natural sources, such as Trinidad Lake asphalt [9]. By 1963, over 90% of the total asphalt and asphalt products sold in the United States was derived from petroleum. From 1923 to 1936, petroleum tars derived from a thermal cracking process were often used in paving and

industrial products. Since 1936, the use of this process has gradually declined.

Few early reports concerning the biologic effects of asphalt or asphalt fumes have been found in the literature. Henry [24] analyzed 3,753 cases of cutaneous epitheliomas reported between 1920 and 1945 to the Chief Inspector of Factories in Britain under the Workmen's Compensation Act. The definition used for reporting these cases was "epitheliomatous ulceration or cancer of the skin due to pitch, tar, bitumen, paraffin or mineral oil or any compound or residue of any of these substances or any product thereof contracted in a factory or workshop." Henry's analysis consisted of the correlations of occupations, suspected causative agents, and sites of the cancers. In only one instance was the cancer attributed to bitumen (asphalt). This instance involved a facial epithelioma on a worker who had been employed for 22 years at an asphalt factory where only natural bitumen was used. No further details were reported.

In a 1949 communication, Hueper [25] reported that the total number of recorded cancers attributed to oil products from all sources was about 100. He stated that this was an "astonishingly" small number considering the large number of persons exposed to oil products, including heavy lubricating oils, fuel oils, paraffin, tars, coke, and asphalt, throughout Europe and the United States. Sources of these data and the basis for the characterization "astonishingly" small were not given.

Other historical studies of occupational carcinogenesis have placed asphalt workers into broad categories like "paviors, street masons, concretors, and asphalters" [26], making it impossible to evaluate the number of cancers attributable to asphalt exposure alone.

Effects on Humans

Reports detailing toxic effects of asphalt on humans are scarce, and attribution of asphalt as the causative agent of these reported effects is often uncertain. Workers who came in contact with asphalt often had had previous contact with coal or tar or simultaneous contact with asphalt and tar, and confusion over the definition of asphalt, as in "asphalt is also obtained by coal distillation..." [14], increases the difficulty of analyzing the literature.

Zeglio [15], in 1950, published observations on 22 workers aged 18-61 years who insulated electrical cables and telegraph and telephone lines for a large Italian company. Medical histories emphasizing respiratory disorders were obtained, and clinical and roentgenographic examinations of the respiratory tract and special nose and throat examinations were performed on each worker. Only bitumens (natural asphalt) were reported to have been used in the insulating process, but Zeglio pointed out that "true bitumen" was often adulterated with residual pitch from coal tar distillation. He did not indicate, however, whether the bitumens used by this company had been so adulterated. The workers were exposed to fumes from tanks containing bitumen heated to 120 degrees (presumed to be degrees Celsius, equivalent to 248 F). Although the concentration of the fumes in the working environment was not measured, Zeglio stated that the atmosphere had an acrid odor that was irritating to the nose and throat and that stimulated coughing.

Workers exposed to the atmosphere of the plant complained of coughing with expectoration, a burning sensation in the throat and chest, and frequent hoarseness. Headache and nasal mucous discharge were also

frequently mentioned by the workers as sequelae of exposure. Workers employed in the plant for only a few months were more aware of these annoying sensations than were those employed for several years, and the disturbances usually diminished rapidly after the employees left work (presumably after the end of a work shift). Even though the subjective sensations appeared less intense in the workers with more seniority, they were at the same time more persistent and involved nasal mucous discharge and inflammation, coughing with expectoration, changes in vocal timbre, and frequent loss of voice. Tonsillitis, pharyngitis, acute febrile bronchitis, and nosebleeds were frequent effects and caused employee absence during the cold seasons (not further defined).

During physical examinations, 10 cases of rhinitis, 13 cases of oropharyngitis, 4 cases of laryngitis, and 19 cases of bronchitis were diagnosed in the 22 workers, with some workers having multiple diagnoses [15]. The mucosal involvement, which varied in severity from simple to chronic atrophic inflammation, was sometimes accompanied by scabby exudates and swelling. The pulmonary signs varied from simple, harsh respirations with some rales to severe pulmonary involvement, including basal hypophonesis with increased tympanic zones from emphysema, thoracic hypomobility, extensively harsh respiration with protracted expiration, and rales and sibili. Roentgenographic examination disclosed an increased vascularization in the larger bronchial areas, particularly evident in the mediobasal zones in all the workers with bronchitis. Those workers with the more intense clinical signs also had bronchiectatic formations. No infiltrative or erosive lesions of the pulmonary parenchyma were observed in any of the workers examined.

Zeglio [15] concluded that a simple characterization of bitumen vapors as "irritating" did not recognize the harmful anatomic and functional changes of the respiratory tract that they caused. He pointed out that the size of his population was small and that individual variations in resistance to bitumen vapors may have been more important than the length of exposure to the vapors in determining the severity of the clinical manifestations. He also pointed out that particular factors predisposing the workers to the damaging effects of bitumen, such as fatigue, smoking, and excessive alcohol consumption, were not considered in this study. Zeglio did not report the source of the bitumen used in the workplace, nor did he differentiate between fumes and vapors, terms which he used interchangeably. The characterization of the atmosphere as acrid and irritating, however, suggests the presence of coal tar pitch volatiles in the workplace [21]. Information concerning the bitumen vapor composition and concentration and the length of exposure necessary for eliciting adverse effects, was not included in this report [15]. Thus, it is not possible with these data to determine the relative hazard ascribable to the asphalt fumes.

Guardascione and Cagetti [16] reported on a case of laryngeal cancer in a worker employed in road bituminization. The worker had been employed by the same firm for 31 years, and his primary jobs had been lighting the furnaces for heating the bituminous liquid (containing asphalt, tar, and pitch) and mixing, transporting, and pouring the liquid on the roadbed. When questioned, the worker complained of occasional skin rashes. He also reported noticing the presence of blackish specks in his sputum, chronic bronchial manifestations, and a transitory dysphonia arising from

laryngitis. Approximately 4 years before the laryngeal cancer was diagnosed, the worker had a complete loss of voice lasting 1 year. A diagnosis of squamous cell carcinoma of the left vocal cord was made from biopsy material, and the worker underwent a total laryngectomy.

Guardascione and Cagetti [16] pointed out the problems involved in determining the causative agent for this carcinoma, especially the fact that bitumens were often considered as the same product whether they were natural, were derived from the distillation residue of petroleum, or were derived from tar pitch. They postulated that, since polynuclear aromatic hydrocarbons are generally absent or found only in trace amounts in bitumens obtained from petroleum residues, the only substances used by this worker that had carcinogenic properties were the tar and tar products; however, data to support their assumptions were not presented. This report, though valuable in pointing out a possible occupational hazard, did not provide enough data to correlate exposure and effect. Guardascione and Cagetti's discussion of the carcinogenic propensity of petroleum residue versus tar indicates that the tar mentioned may have been coal tar, but this is not explicitly stated in the report.

Two reports have dealt indirectly with health hazards from asphalt fumes. A NIOSH health hazard evaluation report [18] on a roofing application project presented results from medical interviews, limited physical examinations, and environmental measurements of polycyclic particulate organic matter (PPOM), polynuclear aromatic hydrocarbons (PAH's), benzo(a)pyrene (BaP), and benzo(e)pyrene (BeP).

Thirty-four employees involved in the application of a roof coating composed of coal tar pitch and asphalt were questioned and examined.

Particular emphasis was placed on determining the circumstances surrounding current and previous work-related skin and eye irritation. The skin and eyes of each worker were examined by an occupational physician. Air samples for examination of employee exposure to total particulates, PPOM, PAH's, BaP, and BeP were collected by personal air samplers attached to the workers' collars. Area air sampling was also conducted close to the machines from which fumes were emitted. The samples were analyzed gravimetrically for total particulates. Ultrasonic vibration of the total particulates followed by extraction by cyclohexane yielded the PPOM, which was then further extracted to obtain PAH's and, finally, analyzed for BaP and BeP by flame ionization gas chromatography. Bulk pitch and asphalt samples were also analyzed for PPOM, PAH's, BaP, and BeP.

Twenty-three (69%) of the examined workers complained of apparent skin photosensitivity, which they attributed to exposure to pitch [18]. Fifty percent of the employees also complained of conjunctival symptoms that they thought were related to exposure to pitch as well. During the physical examinations, 6 (18%) of the workers were observed to have conjunctivitis, 6 (18%) showed evidence of localized skin photosensitization reactions, and 5 (15%) had generalized erythemas that might have been caused by exposure to solar radiation alone. A number of chronic skin and eye disorders were noted during the examinations, but these were not discussed in detail in the report. The concentrations of PPOM in the personal air samples obtained for 26 employees exposed to the pitch volatiles varied from less than 0.02 mg/cu m to a maximum of 0.49 mg/cu m. A statistically significant ($P < 0.05$) correlation was found between exposure to PPOM from the pitch at concentrations greater than 0.2

mg/cu m on the day of examination and the presence of conjunctivitis observed during examination.

Analyses of the bulk pitch used on this job showed 4.89% by weight of PPOM, which in turn contained 1.9-13% PAH's, and 270 ppm (0.027%) of BaP-BeP, whereas analyses of the asphalt showed that it contained 10.3% by weight of PPOM, that 0.5-3.2% of the PPOM was PAH's, and that BaP and BeP were not detected (less than 6 ppm) [18]. The authors concluded that these results indicated a significant difference between the composition of pitch and that of asphalt, with the pitch containing more higher-molecular-weight PAH's than the asphalt.

No attempt was made in this study to differentiate medically between health effects caused by asphalt fumes and those caused by coal tar pitch volatiles; however, employees were asked to list subjective symptoms caused by working with the asphalt [18]. None of the employees questioned described any symptoms that they believed to be related to exposure to asphalt except for occasional burns from contact with the hot asphalt. The authors concluded that there was no evidence that asphalt fumes made an important contribution to the acute eye and skin problems suffered by these roofers.

A similar study, reported by NIOSH [20] in 1976, obtained roofing workers' responses to a questionnaire pertaining to causes of skin and eye irritation. As in the previous study, virtually no irritation was attributed to asphalt by the workers. These studies [18,20] indicate that asphalt fumes are not considered a hazard by employees who work with asphalt. However, no data were reported for possible respiratory effects,

nor was it clear if the questionnaire or physical examination addressed these possible problems.

Epidemiologic Studies

No workplace concentrations of asphalt fumes or exposure conditions of workers were given in the one epidemiologic study found. Subjective data gathered in response to a questionnaire have been reported and are included in this section.

Results of a health survey by Baylor and Weaver [27] of asphalt workers from 7 asphalt companies and a control group from 25 oil refineries were published in 1968. A medical assessment including a physical examination and detailed medical and occupational histories was conducted as completely as possible on each of 462 asphalt workers and 379 refinery workers in a control group. Employees with 5 or more years of work in the asphalt industry were included in the asphalt exposed cohort. The physical examination emphasized the skin and the respiratory tract and included chest roentgenograms. The questionnaire covered number of years in asphalt work, type and geographic source of asphalt used, and smoking and weight histories. Lung cancer had previously been diagnosed in one of the workers in the control group. Skin cancers were reported in the medical histories of two asphalt workers and four workers in the control group. At the time of the survey, skin cancer was present in four asphalt workers, but two of these workers had had skin cancer prior to beginning work in the asphalt operations. The medical histories also reported one carcinoma of the stomach in an asphalt worker and one carcinoma of the colon in a worker in the control group.

Instances of respiratory disease other than cancer were recorded in the medical histories of 31 of 360 asphalt workers (8.6%) and in 12 of 277 control workers (4.3%) [27]. It was not stated whether these past medical histories referred only to time before asphalt exposure. The majority of these cases had been diagnosed as chronic bronchitis, with a few cases of asthma or emphysema. At the time of the physical examination, instances of lung disease were diagnosed in 40 of 462 asphalt workers (8.6%) and in 24 of the 379 control workers (6.3%). None of these illnesses was described as advanced, severe, or incapacitating, and several were diagnosed solely on the basis of increased bronchial markings on the chest roentgenograms. A publication of the National Center for Health Statistics [28] reported a combined incidence of chronic bronchitis, emphysema, and asthma of 11.8% for males, 16 to 65 years old, in the "usually working" category. A comparison of this value with those for the exposed workers yields relative risks which are not significantly different by the normal-deviate test.

The medical histories reported instances of noncarcinogenic skin disease in 37 asphalt workers (10.3) and in 47 of the workers in the control group (17%). During the physical examination, 26 asphalt workers (5.6%) and 20 workers in the control group (5.3%) were found to have skin diseases, mainly localized and transitory dermatitis; none of the cases was considered extensive or severe. Other health problems discovered during the physical examination included hypertension in 27 asphalt workers (5.8%) and 27 control workers (7.1%), peptic ulcer in 12 asphalt workers (2.6%) and 8 control workers (2.1%), heart disease in 17 asphalt workers (3.7%) and 14 control workers (3.7%), and other miscellaneous disorders paralleling what would be expected in the general population. Incidental

findings indicated that the most serious threats to the health of both the asphalt workers and the control workers were obesity and heavy cigarette smoking. The findings indicated that the asphalt workers were not under a significantly greater health hazard than were the control refinery workers.

Additional information was requested from companies involved in highway construction, roofing, trucking, and insurance, and from State Highway Commissions and Boards of Health, to ascertain health problems attributable to occupational exposure to asphalt [27]. Information secured in this way may not be highly reliable. Thirty-one construction or paving companies reported only one case of ill health that could have been attributed to exposure to asphalt in their 11,478 man-years of work experience with it. Of the 15 states that replied to the questionnaire, 13 cited no cases of illness caused by occupational exposure to asphalt (although over 100 million gallons of asphalt per year were used by 1 of the states), 1 state reported 1 case of transitory nasal irritation caused by asphalt fumes, and 1 state reported 14 cases of dermatitis resulting from contact with asphalt; no further details were supplied.

Three large roofing companies employing over 1,100 asphalt workers reported no evidence of ill health attributable to asphalt [27]. The 112 asphalt roofing employees of 1 company averaged over 12 years of experience with asphalt, with 23 workers having over 20 years of experience. Four trucking companies with a total of more than 5,000 drivers exposed to asphalt fumes and dust reported no known cases of lung or skin disease attributable to asphalt. Five large insurance companies responding to the questionnaire, 1 of which serviced over 43 companies whose primary business was asphalt production or use, reported no known cases of compensation

involving occupational exposure to asphalt. Another insurance company reported four claims involving asphalt: one case of headache, one case of silicosis secondary to asphalt, one case of leukoplakia, and one case of dermatitis. Followup details were not available for these claims.

Baylor and Weaver [27] concluded that "...petroleum asphalt cannot rationally be considered a hazardous substance." They did, however, point out that individual predisposition might cause some workers to be subject to health problems promoted by contact with asphalt or its components, and that their study related to the present commercial use of asphalt only. They also stated that their conclusion did not imply that all asphalt is completely nonhazardous under all circumstances.

Because of the lack of detailed results and exposure data in this report, quantitative evaluation of the results is not possible. Information from past medical histories of the current workforce concerning lung disease other than cancer indicates that the relative risk was 1.96 for the asphalt workers compared with that for the controls, which is significant at the $P < 0.05$ level. The relative risk for current lung disease, however, was not significantly different for the two groups, and the lung disease frequency for the asphalt workers was not significantly different from the lung disease frequency for the US working population [28]. Without further information, an accurate assesment of the potential occupational hazard from asphalt fumes is not possible; however, the authors' conclusion that the workers studied were not under an appreciably greater hazard than the controls is reasonable on the basis of the statistics reported.

The human studies reviewed tend to suggest that the occupational health hazard from exposure to asphalt fumes is minimal, although the work of Zeglio [15] indicates that such exposure may be a contributing factor to respiratory irritation and noncarcinogenic diseases in mixed exposure situations.

Animal Toxicity

This section is divided into three parts to facilitate comparison of the animal data with potential workplace exposure to both asphalt fumes and asphalt and to briefly review the literature related to some of the toxic constituents found in some asphalts. Discussion of some of the problems involved in comparing studies of these constituents is also included.

(a) Exposure to Airborne Asphalt Fumes and Aerosols

NIOSH has not been able to find reports of experiments to determine dose-response relationships for asphalt fumes or reports demonstrating a carcinogenic potential for asphalt fumes. Irritations of the eyes and respiratory tract of animals subjected to extreme exposure conditions have been reported, but environmental concentrations of the fumes for these exposures have not been presented.

Observations of the effects of asphalt vapor on the eyes of rabbits were presented by Truc and Fleig [29] in 1913. The asphalts used for these experiments came from the United States and England but were not further identified. The vapors were generated by heating the asphalt in a retort until dense oily vapor was given off. The vapor was then directed onto the eyes of immobilized rabbits. Concentrations of the vapor in the air contacting the eyes and durations and frequencies of the exposures were not

reported. Only minor transient conjunctivitis was noted in the rabbits exposed to asphalt vapors. A slight infiltration of the cornea was sometimes noted after frequent exposures, but this disappeared within several days after the exposures ended. No other signs of toxicity were observed in the rabbits.

Changes in the respiratory tracts of guinea pigs and rats after inhalation of fumes from petroleum asphalt and coal tar pitch were reported in 1960 by Hueper and Payne [19]. Fumes were generated by placing asphalt or coal tar in an evaporating dish in the exposure chamber and heating the dish to 250-275 F (121-135 C) with an electric heater. Cooled air was delivered to the exposure chambers from air-conditioning units adjusted to maintain a chamber temperature of 75 F (24 C). Fumes were generated for 5 hours a day, during which time 10-30 g of the coal tar or 2-10 g of the asphalt were volatilized. The volatilized amounts were replaced daily with equal weights of the parent material, and fresh material was placed in the heating dish once a week. The exposures were carried out on 4 days each week for 2 years. Thirty 2-month-old Sl3 guinea pigs and 65 2-month-old female Bethesda black rats were exposed in each of the two chambers.

Some guinea pigs and rats exposed to the asphalt fumes developed "extensive chronic fibrosing pneumonitis with peribronchial adenomatosis, associated in the rats with squamous cell metaplasia of the bronchial mucosa or of frequently noted bronchiectatic lumens [19]." None of the adverse changes assumed a cancerous character, and they were stated to have been often observed to result from nonspecific chronic pneumonitis or the respiratory or intravenous administration of various other substances (not further defined).

Hueper and Payne [19] stated that these data, in addition to the results of other studies involving the inhalation of various "carcinogenic" dusts, fumes, or vapors, indicated that the respiratory tract of experimental animals appeared to be much more resistant than the human bronchial tree to "inhaled carcinogens," even when the inhaled carcinogens were administered in much higher doses than those encountered by workers under the worst occupational conditions. They also speculated that the degree of exposure of the lung tissue to inhaled carcinogens was usually inadequate for eliciting a carcinogenic response in experimental animals. No experimental data leading to the authors' characterization of asphalt fumes as carcinogenic were presented in this report. This study did, however, demonstrate a significant respiratory hazard from long-term exposure to high concentrations of asphalt fumes.

Estimate of exposure concentrations can be made for this experiment by making assumptions concerning the rates of airflow through the exposure chambers. Indirect calorimetric experiments suggest that this flow through the chamber would probably be in the range of 1-10 cu m/hour. If airflows of either 1 or 10 cu m/hour are assumed and the approximate amount of asphalt volatilized/hour is calculated from the authors' data [19], the possible range of the exposure concentrations becomes 40-2,000 mg/cu m. These calculations indicate that the exposures in this experiment were greater than are likely to be encountered by workers during representative occupational exposures to asphalt fumes. See Chapter IV for further discussion of concentrations of asphalt fumes found in occupational situations.

Studies of exposures of experimental animals to petroleum asphalt aerosol and smoke (containing fumes) were reported by Simmers [30] in 1964. In the first experiment, 10 male and 10 female C57BL mice were exposed to an aerosol of unstated concentration generated from asphalt derived from a California crude oil. The aerosol was generated by discharging an emulsion of asphalt in hot water through a nebulizer into a large chamber. The aerosol was admitted in 2-second pulses for 16 seconds in each minute; this periodic pulsation was adequate to maintain a fog of asphalt in water for the full 30-minute daily exposure. Exposures were carried out 5 days a week for up to 82 weeks. The animals were restrained with their muzzles projecting into the aerosol chamber.

Autopsies were performed on 17 mice after they died or were killed [30]. Three of these had survived 410 exposures and 10 had survived 280 or more exposures. The number of animals surviving fewer than 280 exposures was not reported and could not be determined from the reported data. The last animal was reported to have been killed 16 months and 18 days (approximately 72 weeks) after the experiment began, although this is inconsistent with the author's statement that exposures were made for up to 82 weeks. The alveolar spaces sometimes contained fluid, and patchy regions of emphysema and bronchiolar dilatation were occasionally observed. A few cases of pneumonitis, localized bronchitis along with minimal peribronchiolar round-cell infiltration, and a papillary adenoma were also observed. To show that the asphalt was actually being deposited in the respiratory tract, four mice were exposed to an aerosol of asphalt radiolabeled with I-131 for either 15, 30, 45, or 60 minutes. The trachea and lungs of these animals were then removed, separated, weighed, and

examined for the radioactive iodine. This experiment showed that the asphalt was being deposited in these organs and that its concentration in the trachea was approximately twice as great as that in the lungs.

Another experiment was conducted to determine the effects of asphalt "smoke" (containing fumes) on mice [30]. Six cages, each containing five C57BL mice, were held in an exposure chamber. "Smoke," generated by heating the asphalt to 250 F (121 C) with an electric heater, was forced through the chamber by a fan. The placement of the cages in the exposure chamber was rotated daily to equalize the effects of any concentration gradient of the "smoke" from the inlet and outlet. Animals were exposed from 6 to 7.5 hours daily, 5 days/week, for a maximum of 21 months. During the course of the experiment, 2,236.6 g of asphalt were volatilized to produce the "smoke" that was blown through the chamber. Food was continuously available in three of the cages, but it was removed during the exposures from the other cages. Controls consisted of six nonexposed male mice that were killed at 20 months of age.

In this experiment, 21 of the 30 exposed mice were subjected to necropsy [30]. Nine of these mice survived 401 days of exposure to the asphalt "smoke," and 15 were exposed to the "smoke" for over 300 days. The last animal was killed after 21.2 months. Again, the length of exposure and the time of killing of the last animal are discrepant, but in a possible way in this instance. During the experiment, the inside of the exposure chamber became covered with a yellowish-brown, oily material that had a strong petroleum odor and showed a yellowish-green fluorescence when illuminated with ultraviolet light. The animals whose food was contaminated with this material ate as much and maintained their body

weights as well as the animals eating the uncontaminated food. No gross evidence of gastrointestinal tract tumors was found in the animals eating the contaminated food, although a few gross lesions were observed. When examined microscopically these lesions were judged not to be neoplastic.

Bronchitis with abscess formation, loss of bronchial cilia, epithelial atrophy and fragmentation, necrosis, and flattening of the epithelium, along with pneumonitis, were frequent findings in lungs of the animals that had inhaled asphalt "smoke" [30]. One bronchial adenoma was also reported. Epithelial hyperplasia occurred occasionally, as did emphysema, often associated with focal lung collapse. Large areas of peribronchial round-cell infiltration were common and extreme bronchial dilatation was sometimes observed, but no tumors were reported. In both experiments, the adverse changes noted in the tracheobronchial tree and lungs of the exposed animals were scattered, with normal areas being found in all animals. Some animals were relatively refractory to the "smoke" and aerosol, while others showed advanced adverse changes after relatively few exposures.

Simmers [30] stated that the tracheobronchial and pulmonary changes observed in these experiments closely paralleled those described in other experiments on the respiratory effects of various air pollutants. He suggested that the changes in the tracheobronchial trees and lungs of mice breathing air polluted with polycyclic aromatic hydrocarbons (PAH's) might have been nonspecific phenomena and that the degree of change was dose-dependent. He also indicated that these findings paralleled the observed fact that not every human exposed to air pollutants shows the same adverse reactions. From the data presented, no statement proposing a dose-response

relationship between polycyclic aromatic hydrocarbon content and degree of adverse change can be supported. The adverse effects observed in these animals closely parallel those observed by Hueper and Payne [19] and indicate again that nonspecific respiratory irritant effects are caused by chronic exposure to high concentrations of asphalt fumes.

Exposure concentrations in this experiment can also be estimated by assuming a chamber airflow of 1-10 cu m/hour and calculating the amount of asphalt volatilized/hour. These calculations indicate a range of possible concentrations of 74-929 mg/cu m. As in the previous experiment, these estimates of the exposure concentrations would mean that the animals used in this experiment were subjected to considerably higher total exposures than would be expected to impinge upon humans during a lifetime of work in the asphalt industry.

The adverse respiratory effects noted in response to long-term exposure to high concentrations of asphalt fumes were similar to those elicited by nonspecific respiratory irritants. The character of these adverse effects indicates that these exposure situations produce lesions that may not be totally reversible. Although these reports raise concern about the possible induction of pulmonary cancers in the animals exposed by inhalation to the airborne volatile components of asphalt, the evidence is insufficient to justify the conclusion that fumes or aerosols of asphalt are carcinogenic. No lesions or tumors of the skin were reported for any of the animals exposed to asphalt fumes in these experiments.

(b) Exposure to Asphalt and Asphalt Mixtures

The biologic responses of animals to exposure to asphalt are dependent on the route of exposure, the crude from which the asphalt was derived, and the method of asphalt extraction from that crude source. The adverse effects reported in animals after exposure to asphalt have often been in response to exposure situations that would seem unlikely to occur in the workplace environment.

In 1959, Simmers et al [31] assessed the carcinogenic potential of a mixture of steam-refined and air-blown asphalts from six different samples supplied by southern California refineries. The asphalts were pooled for this experiment, and a portion of this pooled sample was mixed with sufficient benzene to make it fluid enough to be painted on the skins of animals. A second portion of the pooled asphalts was suspended in olive oil to give a 1% emulsion for subcutaneous injection.

Four groups of C57BL mice were used in this study [31]. In Group 1, which consisted of 32 male and 36 female mice, the asphalt-benzene mixture was applied to the skin of the interscapular region twice weekly with a glass stirring rod. Group 2 consisted of 31 male and 32 female mice that were treated similarly to those in Group 1, but with benzene alone. Group 3, consisting of 33 male and 29 female mice, was injected subcutaneously in the interscapular region with 0.2 ml of the asphalt-olive oil suspension twice weekly; after 41 weeks, the frequency of injection was reduced to once a week because the volume of material in the injection region became excessive. Group 4 consisted of 32 male and 28 female mice that were injected with olive oil alone in the same way as those in Group 3.

Development of cancers in Group 1 was preceded by loss of hair at the painting site, dryness and scaling of the skin, and formation of papillomas [31]. Fifty-four weeks elapsed between the initiation of treatment and the appearance of the first of 12 epidermoid carcinomas at the site of painting. Four mice of this group were alive with well-developed papillomas at the time the paper was presented for publication, and one mouse with an especially large papilloma had died. Hair loss and dry and scaling skin were the only signs reported in Group 2. Group 3 developed injection site tumors consisting of one rhabdomyosarcoma and seven fibrosarcomas. The first sarcoma was observed 36 weeks after injection. No evidence of tumor formation was reported for Group 4. No evidence of metastasis was reported in any of the tumor-bearing animals. The authors concluded that pooled asphalt from western US crude oil sources contained a substance or substances that induced formation of tumors at the contact sites when applied to the skin or injected subcutaneously into C57BL mice.

Because exposure concentrations were not given and because results were reported as the total number of malignant growths rather than as the number of animals with malignant growths, it is not possible to determine a dose-response relationship from this report. The report does, however, clearly demonstrate that asphalt mixed with benzene or olive oil can induce neoplasms in mice. Although these were judged to be malignant on the basis of their morphologies, no metastatic lesions were reported. The authors did not address the possible cocarcinogenicity and irritating or promoting effects of the mechanical action and lipolysis of the dilution media, which might affect the tumorigenic potential of the mixture.

Another study published by Simmers [32] in 1965 was designed to test the carcinogenicity of asphalt that had not been mixed with diluent. In this study, a mixture of three samples of air-blown asphalt was used in one series of experiments, and a mixture of three samples of steam-refined asphalt was used in another series of experiments. The two pooled samples differed quantitatively as to the content of each major chemical fraction: the steam-refined asphalts contained 24% asphaltenes, 31% aromatics, 12% saturated compounds, and 33% resins, while the air-blown asphalts contained 41% asphaltenes, 26% aromatics, 11% saturated compounds, and 22% resins. To make asphalt fluid enough for painting and injection, it was heated by a hot water heat-exchange system to temperatures between 70 and 100 C (158-212 F). The temperatures of the asphalts when they were applied to the mice were not reported.

Twenty-five male and 25 female C57BL mice, approximately 6 weeks old, were painted with air-blown asphalt from 1 to 3 times each week, depending on skin condition [32]. The hair over the scapular region was closely clipped before the paintings began, and 75-100 mg of asphalt was applied at each painting. The method of determining the amount of asphalt applied during each painting was not reported. After 7 weeks, 14 males and 18 females from this group survived an epizootic of pneumonitis. The number of paintings for those animals surviving the epizootic ranged from 22 for an animal found dead after 63 days to 270 for a mouse killed after 21 months and 23 days. A similar group of 25 male and 25 female mice of the same age was painted three times a week with steam-refined asphalt. Fifteen males and 12 females from this group survived the epizootic. After 1 year of treatment, one male and five females survived. Causes of

mortality for the other animals were not reported, although all showed unspecified skin reactions to asphalt. Eight male and five female mice were therefore added to this group to complete the study. The number of paintings for the animals from this group on which autopsies were performed ranged from 16 to 240. No controls were reported for either of these groups.

Simmers [32] stated that the abilities of the undiluted asphalts to coat the skin were poor. Because the asphalt hardened into a plaque, which the animals often pulled off along with attached skin, Simmers conducted another set of experiments, using air-blown asphalt diluted with toluene (10:1). Ten male and 10 female mice were painted with this mixture 3 times weekly for 2 years, or for a total of 284 paintings. The asphalt-toluene applications averaged 20-30 mg/painting. To maintain the paintable consistency of the mixture, toluene had to be added periodically because of its rapid evaporation. An exact determination of the asphalt concentration was not possible because of this evaporation and the repeated addition of solvent. Five male and 10 female mice received applications of toluene alone, 3 times a week for 19 months, for a total of 230 applications, and served as controls. The author stated that he did not estimate the amount of toluene applied because of its rapid evaporation.

Dermatitis described by the author as chronic was usually observed at the site of air-blown asphalt painting [32]. One adenoma of the lung, one papilloma at the site of painting, and one tumor of skin accessory-structure origin were found in this group; no carcinomas were reported.

In the group painted with steam-refined asphalt, three animals developed epidermoid carcinomas at the site of application [32]. One of

these was of skin accessory-structure origin. Two papillomas were also found in this group.

In the group painted with air-blown asphalt mixed with toluene, 9 epidermoid cancers, including 1 of skin accessory-structure origin in an animal painted 147 times, were identified [32]. One cancer in an animal painted 240 times involved a regional lymph node, and another animal painted 252 times had a cancer that had invaded the scapula. Two lung adenomas were observed in other animals. In two animals, the only abnormal condition observed was chronic dermatitis.

Tissue samples from six of the toluene controls were examined microscopically, and one of these mice had a small papilloma [32]. All showed loss of hair, scaling, and thickening of the dermis and epidermis. These findings indicated that the general irritant effects observed were probably caused by the solvent used and not by the asphalt. Examination of the toluene showed a faint apple-green fluorescence under ultraviolet light, indicating the presence of contaminants.

In yet another experiment, 25 male and 25 female C57BL mice each were given 1 subcutaneous injection in the interscapular region of 200 mg of steam-refined asphalt [32]. After 111 days, nine males and four females, having no palpable asphalt deposits, were reinjected with another 200 mg/animal of steam-refined asphalt. Another group with the same number of mice was injected with the same dose of air-blown asphalt. After 4 months (120 days), 11 males and 7 females in the second group were reinjected as above.

In the mice injected subcutaneously with steam-refined asphalt, one adenoma of the lung was discovered, but no carcinomas [32]. On autopsy,

the asphalt was found in the abdominal and thoracic cavities, remote from the intended injection site, in 5 of 15 animals. Wherever the asphalt was found, it was covered by a thin, relatively acellular sheath. Twenty-one animals died during the 23 months of the experiment, the most frequent cause of death being pneumonitis.

In the group of mice injected with air-blown asphalt, one female developed a rhabdomyosarcoma primary to the site of asphalt injection and metastatic to the lungs and liver [32]. A second animal also had a rhabdosarcoma at the site of injection, and a third animal developed a carcinoma, apparently arising from skin accessory-structures, that had metastasized to the left lung.

Simmers [32] concluded that the "low yield" of cancers in the groups painted with undiluted asphalts may have been a result of the inability of the asphalt to maintain an intimate skin contact, since cancers were found when the asphalt was mixed with toluene, thereby making it more fluid. He stated that the small number of cancers found in the injected groups was probably because the chemicals present in the asphalts were not extremely carcinogenic. He also speculated that the carcinogenic hazard to workers using molten asphalt would probably be "inconsequential," since prolonged intimate contact with the skin or subcutaneous injection of the substance would be highly unlikely.

Simmers' [32] method of reporting results leaves some confusion as to how many animals actually developed carcinomas, since he reports the total number of carcinomas and not the number of animals with carcinomas; however, a comparison of his summary statements with the data presented in his tables indicates that he was reporting one carcinoma/mouse in this

study. The large increase in carcinomas in the animals receiving asphalt mixed with toluene is an interesting finding. Since the toluene fluoresced, indicating the possible presence of PAH's, the toluene may have contained carcinogenic or cocarcinogenic contaminants, which renders uncertain the interpretation of the data from this part of the study.

Hueper and Payne [19] subjected various experimental animals to skin painting or to intramuscular (im) injection with one of four road asphalts, a roofing asphalt, or coal tar. The animals used for these studies were C57BL mice, Bethesda black rats, and New Zealand rabbits. The four road asphalts were diluted with acetone so that they could be applied more easily. Applications to the nape of the neck were made two times weekly for up to 2 years. Controls for the painting experiments consisted of 200 mice and 60 rats that were colony animals. One road asphalt was applied to 50 male and 50 female mice, and the other 3 road asphalts were each applied to 25 male and 25 female mice. The extent of dilution of the asphalts and the amounts applied were not specified by the authors.

Two skin carcinomas and 2 papillomas were observed in the 250 mice exposed to the 4 road asphalts by skin painting [19]. Four instances of leukemia and one Kupffer-cell sarcoma were also attributed to the action of these asphalts.

The petroleum roofing asphalt was tested for its carcinogenic potential by painting the heated, undiluted asphalt on the napes of the necks of 25 male and 25 female mice twice weekly for a maximum period of 2 years [19]. Following the same schedule, this asphalt was also painted on the insides of the ears and on 2-cm-square shaved areas of the backs of six rabbits. No neoplastic reactions were observed in these animals.

For im injection, each of the asphalts was diluted 1:1 with tricaprylin [19]. Each tricaprylin-asphalt mixture was then injected into the right thigh muscle of a separate group of 50 mice and 30 rats in doses of 0.1 ml for the mice and 0.2 ml for the rats. Injections were given at 2-week intervals, 6 times for the mice and 12 times for the rats. Subcutaneous injections of tricaprylin, amount and schedule unspecified, were given to 144 mice that were used as controls.

In the injected animals, 3 sarcomas were seen at the site of injection in 200 mice and 13 sarcomas at the site of injection in 120 rats [19]. Other sarcomas of the liver and ileocecal lymph nodes and carcinomas of the uterus seen in the experimental groups were the same (not further defined) as those in diluent controls.

A coal tar pitch distillate was obtained by heating coal tar to 250-275 F (121-135 C), collecting the fumes on glass fiber filters, and extracting them with dichloromethane, benzene, and acetone [19]. After evaporation of the solvents, the extract was diluted with olive oil in a ratio of 1:3. Fifty male and 50 female mice, about 8 weeks old, each received 6 injections of 0.15 ml of this mixture at 2-week intervals into the right thigh muscle. Twenty-five male and 25 female mice had the original coal tar, heated sufficiently to make it fluid, applied to the nape of the neck twice weekly for a maximum of 2 years. The tumor incidence for the group of animals receiving applications of asphalt was 4.0%, while incidence for the group of animals receiving applications of coal tar pitch or coal tar pitch distillate was 50.7%.

The painting of coal tar on the skins of 50 mice resulted in 22 carcinomas and 4 papillomas, and the im injection of the coal tar

distillate extract resulted in 50 injection site sarcomas in 100 mice [19].

Hueper and Payne [19] concluded that the road asphalts tested were mildly to moderately carcinogenic for the skin and soft tissues of mice and rats and that occupational and environmental exposure to petroleum road asphalt "may be associated with cancer hazards to the tissues of contact." The asphalts tested, however, were mixed with solvents not used in the asphalt industry. Under these exposure conditions, 2.8% of the 250 mice painted with road asphalt and 1.5% of the 200 mice injected with road asphalt exhibited malignant growths attributed to the asphalt, and the roofing asphalt tested did not induce any malignant growths. While there is adequate demonstration of a carcinogenic hazard to the animals in this study, it is not clear that an occupational carcinogenic hazard from asphalt should be inferred from the experiment.

The carcinogenic properties of six grades of petroleum residues derived from Russian crude oil sources and of coal tar pitch were studied by Kireeva [17]. A 40% solution of petroleum tar, asphalt, or coal tar pitch in benzene was painted on white, strain SS-57 mice once a week for periods up to 19 months. Applications were discontinued at the appearance of toxic effects or marked weakening of the animals. The dose delivered at each application was not reported. Animals that survived the application period received approximately 70 applications. Microscopic examinations were performed on the tissues of most of the animals.

Four asphalts were tested [17]. For one asphalt, 2 of 50 animals developed skin tumors, the first tumor appearing in the 16th month; one animal had a squamous cell carcinoma and one had a sebaceous adenoma. Pulmonary adenomas and adenocarcinomas were present in five mice of this

series. Another asphalt painted on a group of 50 mice resulted in 1 subcutaneous fibrosarcoma, 1 papilloma, 1 pulmonary adenoma, 2 lymphoreticular sarcomas, and 1 hepatic hemangioma. The first skin tumor appeared during the 12th month, but regressed during the 13th month. The first persistent skin tumor appeared in the 16th month, and 4.6% of the animals eventually developed skin tumors. In a group of 40 mice exposed to a third asphalt, no skin tumors developed, but a pulmonary adenoma appeared in 1 animal. One pulmonary adenoma and 1 skin tumor that regressed were found in 37 mice exposed to a 4th asphalt. The 23 animals in the control group, painted with benzene alone, developed no skin tumors, although 1 animal developed pulmonary adenomas.

Fifty-two mice were tested with a petroleum tar [17]. The first skin tumor appeared at 9 months, and 18.4% of the animals alive at this time eventually developed tumors. Of the nine mice with tumors, five had squamous cell carcinomas, one had a fibrosarcoma, and three had papillomas of the skin. Pulmonary adenomas and adenocarcinomas also occurred in seven of these mice, and two cases of unspecified systemic lesions were reported.

Forty-seven mice were tested with another petroleum tar [17]. Tumors developed in 9.5% of the 42 mice from this group that were alive at the time of the first tumor appearance at 10 months. Of the four mice with tumors, two had papillomas and two had squamous cell carcinomas. In addition, all four animals had primary pulmonary adenomas.

Coal tar pitch was applied to the skins of 49 mice [17]. The hair was lost from the site of painting after the first application, and the first tumor appeared after 3 months. The majority of the animals developed multiple tumors, both small papillomas and large cornified disintegrating

tumors. Skin tumors, mainly squamous cell carcinomas with marked keratinization, were found in 86% of these mice. A few animals had infiltrative nonkeratinized foci with markedly atypical cells. Pulmonary adenomas were identified in eight mice, and one mouse had a squamous cell carcinoma of the stomach. The malignant tumors had a marked tendency to metastasize.

The mice receiving applications of petroleum tar or asphalt lost the hair at the site of painting more gradually than those mice painted with coal tar pitch, and their hair gradually grew back during the course of the experiment [17]. During the first 4-5 months, these animals lost weight, were sluggish, and had a marked thinning of the coat.

Nontumorigenic skin effects were observed in both the control and the experimental groups [17]. These effects included epidermal atrophy, focal hyperplasia, and atrophy of the hair follicles. Atrophy of the sebaceous glands in the controls and partial or complete atrophy of the skin papillae and hyperkeratosis with acute and chronic inflammation in the experimental animals were also reported.

Kireeva [17] concluded that the blastomogenic properties of asphalts were considerably weaker than those of coal tar pitch. Kireeva also stated that there was a definite correlation between the benzo(a)pyrene content of the substances tested and their carcinogenic activity, but no analytical data were included in the report to support this statement. The work of Kireeva clearly demonstrated different carcinogenic activities for the benzene solutions of coal tar pitch, petroleum tar, and asphalt used in this study. Two of 177 animals (1.1%) painted with the asphalt-benzene mixture developed malignant skin tumors, whereas 7.1% of the animals

painted with petroleum tar and 86% of those painted with coal tar pitch responded in this way.

Wallcave et al [3] attempted to correlate the PAH contents of eight asphalts and two coal tar pitches with the extent of tumor induction in mice whose backs were painted with these substances. Chemical analyses and separations of the PAH fractions of the asphalts and coal tar pitches were carried out by extraction of the parent materials and subsequent chromatography to produce an "interference-free" PAH fraction. A concurrent test column with known amounts of four PAH's was used as a control.

An estimate of recovery efficiency was made by adding benzo(a)pyrene (BaP) to the asphalt and then analyzing by the above procedure [3]. It was determined that recovery was approximately 65%. BaP concentrations in the asphalts ranged from "not detected" to 27 ppm for the unsubstituted compound and from "not detected" to 69 ppm for the alkyl derivatives. Benz(a)anthracene ranged from "not detected" to 35 ppm for the unsubstituted compound and from 0.05 to 109 ppm for the alkyl derivatives. Other PAH's were present in the same range of concentrations. The BaP concentrations in the two coal tar pitches were 0.84 and 1.25%, and the benz(a)anthracene concentrations were 0.89 and 1.25%. A larger number of PAH's were identified in the coal tar pitches, and each PAH was present in much higher concentrations in the pitches than in the asphalts. For skin application, the asphalts were dissolved in benzene to form 10% solutions and the coal tar pitches to form 9% solutions with respect to the PAH content of the parent material.

Random-bred Swiss albino mice, 7-11 weeks old, with average weights of 20 g for female and 25 g for males, were used in the study [3]. A patch of hair approximately 1-inch (2.5 cm) square was shaved from the back of each animal at the beginning of each experiment and was kept nearly free of hair during the experiment by periodic clipping with scissors. Twenty-five microliters of each material, containing PAH's equivalent to approximately 2.5 mg of asphalt or 2.2 mg of pitch, was applied to the shaved area twice a week for an average of 81 weeks for the asphalt-exposed animals and 31 weeks for the pitch-exposed animals. Each asphalt or pitch was applied to a separate group of animals that ranged in size from 24 to 32 animals at the time of autopsy. A control group of animals was painted with benzene alone twice a week. Autopsies were made on a total of 218 animals exposed to asphalt, 58 exposed to coal tar, and 26 painted with benzene. Skin sections and all grossly abnormal organs were studied microscopically in order to characterize the tumors.

Abnormal features observed in the mice to which either asphalt or coal tar pitch was applied were epidermal hyperplasia, inflammatory infiltration of the dermis, and occasional ulceration and abscess formations [3]. Amyloid accumulations in the spleen and kidneys were frequent in animals painted with asphalt. The authors did not expand on this observation. Carcinomas of the breast, adenomas of the lungs, malignant lymphomas, osteomas, stomach papillomas, endometrial carcinomas, mesotheliomas, fibrosarcomas, and malignant schwannomas were consistently found in both exposed and control groups and their character and frequency were not significantly different. Skin tumors were found in 6 of 218 asphalt-painted animals, and 1 of these was classified as a carcinoma.

Fifty-three of 58 animals (91.4%) that received applications of coal tar pitch developed 84 skin tumors, 31 of which were classified as carcinomas. One of 26 control animals developed a papilloma.

Wallcave et al [3] concluded that the strong tumorigenic activity of coal tar pitch was "in accordance" with the high PAH content of this material, but that the number of tumors in the asphalt-painted group was too small to permit conclusions about the relationship of the tumorigenicity of asphalt to its PAH content. They suggested that the differences in carcinogenicity between the asphalts and coal tar pitches could result from the different methods of production for the two classes of materials, especially the temperatures involved in their manufacture. The maximum temperatures involved in asphalt production were reported as 350-400 C (662-752 F) and the temperatures generating coal tar pitch were reported as having been in excess of 1,000 C (1,832 F).

The different carcinogenic potentials of asphalts and coal tar pitches is well supported by the experimental data, but the hypothesis presented for this differential carcinogenicity (difference in manufacture) is not based on the data obtained from this experiment and is probably only a partial explanation. The differing sources for asphalt and tar (petroleum and coal, respectively) certainly contribute also to different compositions of these products.

In each of these papers, the adverse effects reported have been in response to exposures that are unlikely to be faced by US workers. Some of the studies with asphalts have involved solvents not used in the asphalt industry, and the exposure schedules have not included washing between subsequent applications. Even under these exposure conditions,

carcinogenic effects were observed in relatively few of the experimental animals, and the authors' estimations of the hazards of contact with asphalt have been contradictory [3,17,19,31,32].

(c) Exposure to Asphalt Constituents

Studies [11,33] have implicated some asphalt constituents as presenting carcinogenic hazards. Although these studies cannot realistically be used to develop an environmental concentration limit for asphalt fumes, they are included in this document in order to represent fully the types of experiments that have been conducted to determine asphalt toxicity.

In 1965, Simmers [11] described the carcinogenic responses elicited in mice by the combined saturated and aromatic fractions of a steam-refined asphalt. The parent asphalt was divided into four fractions (asphaltenes, aromatics, resins, and saturated compounds). The procedures for separation of the asphalt fractions involved dissolving the asphalt in benzene followed by precipitation of the asphaltenes with n-pentane, filtration, absorption of resins and polar compounds on clay in a recycling column, absorption of the aromatic and saturated constituents on an alumina-silica gel column, and elution of the saturated components with pentane and of the aromatic ones with benzene. Examination by ultraviolet fluorescence of the four fractions showed that most of the fluorescent activity was in the saturated and aromatic fractions. A mixture of the saturated and aromatic components (freed of solvent) was used in this study.

Twenty-five male and 25 female C57BL mice 20-22 weeks old were used in this study [11]. The mixture of the two fractions was rubbed into the fur of the interscapular area with a glass stirring rod three times a week.

The weight of each application was determined to be approximately 33.4 mg by making 10 separate applications on a dried, weighed mouse pelt and then reweighing the pelt.

Autopsies were performed on 40 of the original 50 animals but 10 of these were not examined microscopically because they showed no gross evidence of neoplastic formations [11]. The minimum number of paintings was 72 and the maximum number was 242. Eleven animals survived the maximum number of paintings. Hair loss, along with dry and scaly skin, was a consistent finding in all animals.

Thirteen of the 30 mice that were studied microscopically had cancers, including 7 epidermoid carcinomas, 5 basal cell or basosquamous carcinomas, and 1 leiomyosarcoma underlying a papilloma [11]. In addition, one animal had an epidermoid carcinoma of the anus and a leiomyosarcoma of the small intestine, and another one had a carcinoma probably of sebaceous gland origin. In one of the animals, an epidermoid carcinoma had metastasized to the lung.

Simmers [11] concluded that, since the aromatic-saturated asphalt fractions tested had a much greater carcinogenic potential than the parent asphalt that he had previously tested [32], the carcinogenic activity of asphalt probably resided in these fractions and was possibly due to the PAH's in them. Simmers' conclusions are reasonable, based on these data and his previous work, but testing for carcinogenic activity of the other fractions and the determination of dose-response relationships are necessary to confirm them.

In a further study, Simmers [33] reported on the effects of subcutaneous injection of the saturated and aromatic fractions of asphalt

into mice. The mixture was obtained in the same manner described previously [11]. In the first experiment, 20 male and 27 female C57BL mice, 9 weeks old, were injected once interscapularly with 0.5 ml of the asphaltic material. In the second experiment, 26 male and 23 female mice were injected with 0.25 ml of the material every other week for 16 weeks, making a total injected volume of 2 ml/animal. A third experiment began with 12 male and 16 female mice 2.5-3 months old. One-milliliter injections were given to these mice for an average total dose of 9.7 ml/animal. The maximal number of injections, 11 during 13.5 months, was given to 8 mice. Three females escaped and 9 males died by the 15th day of this experiment because of an experimental error, and these mice were replaced by 11 male mice, 5.5 months old.

In the first experiment, autopsies were made on 36 mice and tumors were observed in 8 of them [33]. Four adenomas were found, along with four adenocarcinomas of skin accessory-structure origin. One instance of lymphatic leukemia was also reported. In the second experiment, autopsies were made on 39 mice and 7 tumors were reported; these included 1 adenoma of the lung, 2 leiomyosarcomas, 1 adenocarcinoma of the skin accessory structures metastatic to the liver, 1 adenocarcinoma of the lung, 1 fibrosarcoma, and 1 infiltrating lymphoma or small-cell carcinoma of the lung. In the third group of mice, autopsies of 19 animals revealed 7 tumors, including 4 adenocarcinomas and 2 adenomas. One animal in this group had lymphocytic leukemia. A total of 22 tumors were found in these experiments and 15 were classed as malignant. The average survival times of the mice that were studied microscopically were 17 months for the first group, 16.5 for the second, and 13.4 for the third.

Simmers [33] noted that there was relatively little difference between the first and second groups, which received total asphalt fraction doses of 0.5 and 2 ml/animal, respectively, but that there was a large difference between these two groups and the third group, which received an average of 9.7 ml/animal, both in length of survival and in percentage of tumors found during autopsies of the animals. He stated that these observations supported the hypotheses that the tumorigenic effects were dose-related and that the tumorigenic properties of hydrocarbons were more dependent on the dose administered than on the length of exposure. Although NIOSH does not agree entirely with the author's choice of adjective, it does agree that there is a difference between the third group and the other two in tumorigenesis (7 found versus 4 expected on the basis of the combined incidence in the other two groups) and length of survival (13.4 months versus the minimum of 15.7 months expected on the basis of the combined durations of life in the other two groups).

The carcinogenic potential demonstrated in the two previous studies indicates that there is a hazard from the saturated-aromatic fraction of asphalt, although animal studies with undiluted asphalt, as well as reports of human exposure studies [18,20,27] previously discussed, indicate that this hazard is minimal under workplace conditions. The literature [19,32] indicates that the asphalt constituents responsible for inducing neoplastic growths are present only in amounts that are not likely to induce neoplasias, and that only by fractionating or solubilizing the asphalt with specific solvents can a concentration of these potential carcinogenic constituents be obtained that is high enough to increase significantly the probability of malignant growths.

Comprehensive reviews of the complexities of the carcinogenic response are available from several sources [34-36]. Carcinogenic chemicals can be categorized as either direct carcinogens or procarcinogens [36]. The procarcinogen group contains the PAH's. The PAH's are metabolized by specific enzyme systems to ultimately yield epoxides, which are thought to be the active carcinogens. Conversely, enzymes such as epoxy hydase and glutathione transferase detoxify these epoxides, so that the probability of cancer induction in an animal after exposure to the procarcinogen is a resultant of the relative rates at which the activating and detoxifying systems are able to operate.

The PAH epoxides may exert their carcinogenic potential as strong alkylating agents, which can alkylate the DNA or RNA bases or a variety of nucleophilic groups, such as sulfhydryl, carboxyl imino, or phosphoro. A covalent binding to DNA bases has also been observed with the benzpyrenes and other polycyclic hydrocarbons; this system requires activation by radiant energy or through free radical formation.

The total amount of alkylation or covalent binding is dependent on the initial concentrations of a carcinogenic substance. Since other factors also influence the expression of the carcinogenic potential, eg, detoxification reactions and DNA repair, "trace" quantities of these carcinogens have a very low probability of eliciting a tumor. Not enough information is currently available to determine the rates and probabilities of the various competing reactions in vivo. Further, there is no information that indicates the extent of somatic mutation that would justify prediction of a greater carcinogenic influence than that which occurs naturally.

The carcinogenicity of various PAH's is generally accepted on the basis of animal experiments [37-39]. Several of these PAH's have been found in petroleum asphalt and have been implicated as the causative agents for the neoplastic reactions caused by asphalt in animal experiments [3,17]. Especially well documented is the case for the carcinogenicity of benzo(a)pyrene (BaP) [37-40]. The maximum reported BaP content of asphalts from different crude sources were: 0.6 ppm in an asphalt from a Russian crude oil [41]; nondetectable (less than 6 ppm) from a US roofing asphalt [18]; 0-27 ppm in eight US paving asphalts [3]; and 11, 14, and 22 $\mu\text{g}/1,000 \text{ cu m}$ (1.1, 1.4, and 2.1×10^6 ppm; * means "to the negative power of") in the asphalt fumes from two US paving plants [5]. Two emission studies reported no detectable BaP in emissions from asphalt kettles [42], and BaP as approximately 0.00003% of the total particulate emissions from saturant plants and 0.00008-0.00019% of the total particulate emissions from air-blowing facilities [43]. In comparison, the concentrations of airborne BaP for January through March 1959 from 74 American cities in 49 states, the District of Columbia, and Puerto Rico ranged from 0.11 to 61 $\mu\text{g}/1,000 \text{ cu m}$ (0.01 to 6.1×10^6 ppm) with an average of approximately 11 $\mu\text{g}/1,000 \text{ cu m}$ (1.1×10^6 ppm) [44]. These values indicate that the BaP concentrations reported in asphalt fumes [5] are no higher than background concentrations found in many American cities. Other PAH's in various asphalts have been reported as being in the same range as the BaP [3]. Although data of this type suggest a possible carcinogenic potential for asphalt and asphalt fumes because of the presence of a known carcinogen, they also indicate that the hazard faced by asphalt workers is of the same order of magnitude as that faced by the general population exposed to urban air.

The presence of known carcinogenic chemicals in some asphalts is a cause for concern. The occupational health hazard faced by asphalt workers is difficult to assess, however, on the basis of the presence of these chemicals alone, because of the interactions of various PAH's and the dependence of tumor yield on experimental procedure.

Hieger [45] reported the results of experiments performed to demonstrate the dependence of tumor yield on factors that are generally ignored when comparing the results from different experiments dealing with carcinogenic substances. These factors, such as the number of applications, volume of injected material, and potentiation and inhibition of carcinogenesis within the same class of chemicals, are of extreme importance in trying to correlate the results of several separate experiments to determine concentrations for persons working in a complex environment that will have minimal, if any, impact on the worker's health. Hieger's experiments were designed to investigate the effects of (1) the frequency of application of a carcinogenic substance, (2) the vehicle used for injection of a carcinogen, and (3) the volume of injected material on tumor formation, and to determine minimal doses for tumor formation for single subcutaneous injections of dimethylbenzanthracene and BaP.

The interscapular skins of 75 C57BL mice were painted with a 0.75% solution of BaP in benzene/liquid paraffin (9:1) at the following intervals: once at the beginning of the experiment, once after 8 weeks, and once after 20 weeks [45]. Of the 75 mice, only 2 developed papillomas. Another 75 mice were painted with a similar solution containing 0.25% BaP once every 2 weeks for 30 weeks, increasing the number of applications over the first group but maintaining the same total dose delivered. This group

developed 25 papillomas, 20 of which developed into epitheliomas.

Single subcutaneous injections of 20 μ g of BaP in 0.2 ml of olive oil yielded 22 injection-site sarcomas in 65 mice (33.8%) of the Swiss, F2, and C57BL strains [45]. Seventy-five mice similarly injected with the same solution containing an addition of 10% cholesterol developed 27 sarcomas (36.0%). Addition of 66% of stearic acid to the original solution of BaP in olive oil resulted in single injections producing only 10 sarcomas among 59 mice (16.9%). The difference between the results in the first two groups is not statistically significant, but the incidence of sarcomas in the third group is clearly lower than that in the other two.

Seventy-five mice of the C57BL, Swiss, F2, and stock strains were given single subcutaneous injections of 20 μ g of BaP in olive oil as in the previous experiment, but in a volume of 0.01 ml instead of 0.2 ml [45]. In this case 4 animals (5.3%) developed tumors as compared with 22 (33.8%) in the previous experiment. Forty-five C57BL, BALB, and stock mice were then given injections of 0.02 ml of BaP solution at 10 different sites, for a total injected volume of 0.2 ml (20 μ g of BaP). Two animals (4.4%) developed tumors.

Single subcutaneous injections of dimethylbenzanthracene and BaP into mice of the C57BL, BRO, BALB, Buffalo, and stock strains showed the threshold dose for injection site sarcoma formation to be 1.25-5 μ g/0.2 ml for dimethylbenzanthracene and 1-8 μ g/0.2 ml for BaP [45].

Hieger [45] demonstrated that 15 applications of a carcinogen resulted in more tumors than did 3 applications of the same carcinogen even though the total dose applied was the same. He also showed that the vehicle for injection of a carcinogen could affect the tumor incidence and

that small volumes of injected material (0.01 ml) would result in fewer tumors than larger volumes would (0.2 ml) even though the total doses of carcinogen delivered were the same. Finally, he demonstrated that both dimethylbenzanthracene and BaP have finite no-observable-effect doses for production of neoplasias.

Several other reports have indicated a no-observable-effect concentration for BaP using different animals and routes of administration. In a study using 180 young adult male Syrian golden hamsters, Feron et al [46] reported the results of weekly intratracheal instillations of BaP. Each instillation consisted of 0.2 ml of 9% saline solution in which 0, 0.0625, 0.125, 0.25, 0.5, or 1.0 mg of BaP had been suspended. Doses at each of these concentrations were instilled into 30 animals weekly for a period of 52 weeks. The experiment was terminated after 78 weeks, and autopsies were performed on the animals. Carcinomas were observed only in the animals that received the two highest total doses. A dose-response relationship was demonstrated for nonmalignant tumor formation, and 3 of 30 animals had such tumors at the lowest total dose of BaP, 3.25 mg/animal. Papillomas of the trachea and adenomas of the bronchioles and alveoli were the only tumors reported in the four lowest dose groups. The extent and complexity of the tumors were much greater in the two highest dose groups, and the carcinomas were limited to these two groups. Nonneoplastic reactions included hyperplasia and squamous metaplasia of the tracheobronchial epithelium, alveolar metaplasia, and adenomatoid peribronchiolar lesions.

A report by Payne and Hueper [47] indicated that 12 monthly subcutaneous injections of BaP into C57BL mice were more effective in

eliciting tumor formation than was a single injection of the same total dose. However, this fact became apparent only at total doses above 0.1 mg. At total doses of 0.05 mg, both groups had the same incidence of tumor formation, 10%, after 2 years. At total doses of 0.025 mg, 10% of the single-injection group showed tumor formation. At either a single injection of 0.008 mg, or 12 monthly injections for a total dose of 0.013 mg, none of the animals developed tumors within 2 years. The authors concluded that low-level recurring exposures to a carcinogen were more hazardous than was a single exposure to the same total amount. From the data reported, this conclusion would only be valid for doses above a certain value, approximately 0.1 mg in these experiments.

Poel and Kammer [48] reported tumor formation in C57BL mice after 75 applications of BaP at 1 μ g/animal were applied to the shaved interscapular area 3 times weekly. Neither incidence of tumors nor the differential classification of the tumors as either carcinogenic or noncarcinogenic was reported in the paper. The authors concluded that, at doses as low as 1 μ g/animal, BaP was a tumorigen and that the threshold concentration for tumor induction must be some value lower than this dose.

In a later paper, Poel [49] reported that single applications of less than 376 μ g BaP to the skin of C57BL mice produced only transient papillomas, and only at doses of 752 μ g/animal and greater were epidermoid carcinomas observed. In another series of experiments involving repeated applications of 19 μ g of BaP to the shaved interscapular skin of mice, 1 exposure elicited no tumors in 8 mice, 10 exposures elicited 5 papillomas in 8 mice, 20 exposures elicited 7 papillomas in 8 mice, and 72-117 exposures were necessary before any epidermoid carcinomas were observed.

At the lowest dose tested, 0.15 μg of BaP/animal, 5 of 55 animals showed induced nonmalignant tumors between 42 and 62 weeks after the initiation of exposure. None of these had developed into a malignant tumor by the 75th week, which was the end of the experiment. Poel suggested that BaP at a dose of 0.15 μg /animal administered 3 times weekly could be considered a "threshold level" for carcinogenesis, producing only transient or noninvasive tumors in a minority of the animals and not significantly affecting their lifespans.

Solutions of BaP in acetone with concentrations varying from 0.0001 to 0.5% were painted over the entire backs of Swiss, CAF1, and C57BL mice three times a week for 7-24 months [50]. Concentrations that did not elicit papillomas or carcinomas were 0.001% in Swiss mice, 0.0005% in CAF1 mice, and 0.001% in C57BL mice. The authors stated that "there is obviously a level below which even lifelong exposure to a given amount of carcinogen will not produce tumors in the experimental animal." The lack of determination of the amount of solution or BaP applied during each skin painting makes calculation of the no-observable-effect dose impossible.

In addition to studies showing the carcinogenicity of single PAH's and complex mixtures of these hydrocarbons, several studies have indicated the complexity of the carcinogenic response in experimental animals. Scribner [51] reported the tumorigenic effects of the PAH's phenanthrene, pyrene, picene, benzo(b)chrysene, and anthanthrene, after tumor promotion with 12-O-tetradecanoylphorbol-13-acetate. These PAH's had formerly been thought to be nontumorigenic. He found also that benzo(e)pyrene and anthracene, whose tumorigenicity had been disputed, both induced papillomas, benzo(e)pyrene being more tumorigenic than anthracene under the

conditions of this experiment. Slaga et al [52] also showed the tumor-initiating capacity of benz(a)anthracene following promotion with 12-O-tetradecanoylphorbol-13-acetate.

Steiner and Falk [53] showed that 1,2-benzanthracene and chrysene were carcinogenic, and that their tumorigenic capacities were potentiated when they were injected together. When 1:2,5:6-dibenzanthracene was injected with 1,2-benzanthracene, however, there was apparent inhibition of carcinogenicity, indicating a possible antagonizing effect.

Benzo(a)pyrene has also been shown to be mutagenic, inducing lethal mutations on the X-chromosome in *Drosophila* males [54,55] and causing frameshift histidine-revertant induction in *Salmonella typhimurium* [56]. It has transformed normal hamster embryo cells in culture, causing a hereditary random pattern of growth, the ability to grow continuously in culture and to develop progressively as tumors after subcutaneous inoculation into adult hamsters [57]. Benzo(a)pyrene has also been shown to be mutagenic by the mouse dominant-lethal test [58].

An experiment in which mice were given access to 1 mg of BaP/g of food for their entire life spans, however, showed no effects on fertility or on the gross morphologies of their offspring [59]. This study indicates that ingestion of BaP caused no teratologic effects and no visibly detectable mutagenic alteration and thus draws attention to the importance of route of exposure and animal system used in eliciting a response.

These reports on the carcinogenic and mutagenic potentials of selected polynuclear aromatic hydrocarbons are cause for concern as to the adequacy of any recommendation of finite environmental limits for worker exposure to mixtures containing them. Because of the multiple factors

affecting carcinogenic potency demonstrated by these and many other reports, it is not feasible to attempt to predict from the results the magnitude of the potential occupational health hazard faced by workers exposed to mixtures, such as asphalt, which contain small percentages of these compounds. While the absence of human data and the paucity of reliable animal data demonstrating adverse effects should not be taken to justify the assumption that any mixture containing known hazardous chemicals is completely safe, this situation in the case of a mixture as widely used as asphalt does suggest that the hazard to health from occupational exposure to asphalt or asphalt fumes is minimal. No reports suggesting mutagenic or teratogenic effects of asphalt or asphalt fumes have been found in the literature.

Correlation of Exposure and Effect

Studies correlating biologic effects of asphalt fumes with exposure in workplace environments have not been found by NIOSH, and reports detailing exposure and effects in animals are not abundant.

Irritation of the mucous membranes of the respiratory tract was reported in 22 workers in an insulating company in Italy [15]. Symptoms and signs in these workers, who were exposed to fumes from bitumens heated to 120 (presumed to be degrees Celsius, equivalent to 248 F), included burning sensations in the nose, throat, and chest, nasal mucous discharge, coughing, expectoration, and loss of voice. These exposures were at a concentration described to have been associated with an acrid odor and to have been irritating to the roof of the mouth. The determination of asphalt fumes as the causative agent in this report was suspect since the

authors stated that true bitumen was often adulterated with residual pitch. In another questionnaire study, 34 roofing workers complained of occasional thermal burns from contact with hot asphalt; they stated their belief that they experienced no respiratory effects or skin effects other than the burns when they worked with asphalt, and the authors concluded that asphalt fumes could not be seriously implicated as causes of certain acute eye and skin problems among these men. Seventeen other roofing workers attributed no adverse skin and eye effects to work with asphalt [20]. Two state boards of health, replying to a questionnaire [27], reported 1 case of transitory nasal irritation and 14 cases of dermatitis resulting from exposure to asphalt, but no exposure details were given. Thirteen other state boards of health reported no adverse effects attributed to exposure to asphalt. Thirty-one construction and paving companies with a total of 11,478 man-years of worker experience mentioned only one case of ill health (details not given) said to be attributable to asphalt [27]. Three roofing companies employing over 1,100 workers claimed that there was no evidence of ill health attributed to asphalt. A health survey [27] of 462 oil refinery asphalt workers indicated no significant health problems when compared with a cohort control group of 379 other refinery workers. Comparison of lung disease data from the asphalt workers with statistics on lung disease in the US working population also indicates no significant difference [28]. These reports indicate that asphalt and asphalt fumes constitute a minimal occupational health hazard under present commercial usage conditions in the United States, considering the large number of workers exposed.

Animals exposed to asphalt fumes at high concentrations showed effects similar to those experienced by the Italian workers [15]. Thirty guinea pigs and 65 rats exposed 5 hours/day, 4 days/week, for 2 years, to the fumes generated by volatilizing 0.4-2 g asphalt/hour developed pneumonitis with dilatation of the bronchi, peribronchial adenomatosis, and squamous cell metaplasia of the bronchial mucosa [19]. Thirty mice similarly exposed for 6-7.5 hours/day, 5 days/week, for 21 months, to the fumes generated by volatilizing 0.74-0.93 g asphalt/hour and 20 mice exposed to an asphalt aerosol showed similar effects, including epithelial atrophy, abscess formation, and emphysema with focal lung collapse [30]. Assuming an airflow through the exposure chamber in each of these experiments on the order of 1-10 cu m/hour, the air concentrations of the asphalt volatiles can be estimated to be in the range of 40-2,000 mg/cu m. In both of these experiments, the effects observed were patchy and were clinically similar to those produced by the inhalation of any chemical irritant, with some animals being refractory to the irritant actions of asphalt fumes. Blowing asphalt fumes directly onto the eyes of rabbits caused conjunctivitis and a slight infiltration of the cornea [29]. These conditions were transient and disappeared after the exposure was discontinued.

A dose-effect relationship between exposure to asphalt fumes and the respiratory and eye irritation observed in the animals cannot be determined because exposure information was not reported. These reports do indicate that asphalt fumes have a potential respiratory irritant effect at concentrations estimated to be between 40 and 2,000 mg/cu m.

Carcinogenicity, Mutagenicity, Teratogenicity, and Effects on Reproduction

Two studies [16,24] examined asphalt as a possible human carcinogen. One study [24] reported one case of cutaneous epithelioma caused by asphalt out of 3,753 cases reported to have been caused by other substances. The other study [16] suggested that previous or concomitant exposures to other carcinogenic substances, such as coal tar pitch, may have been the confounding factors.

After dermal application of asphalt alone, not mixed with a solvent, 3 malignant and 4 nonmalignant tumors were observed in 122 mice and no tumors were seen in 6 rabbits [19,32]. The incidence of neoplasms, both malignant and nonmalignant, ranged from zero to 9.5% in four separate experimental populations.

Dermal application of asphalt in a diluent on animals caused the same types of nonneoplastic reactions as did application of the solvent alone, including hair loss, chronic dermatitis, and dryness and scaling of the skin at the application sites [3,11,17,31,32]. Neoplasms, both malignant and nonmalignant, were reported for animals painted with a solution of asphalt with from 2.75 to 25% of the animals in the experimental populations developing neoplasms. A control group of animals in one experiment had a 3.8% incidence of spontaneous tumor formation [3]. When a mixture of only the aromatic and saturate fractions of asphalt was painted on another group of animals, 32.5% of the animals developed neoplasms [11]. These results indicated that under these experimental conditions, asphalt had the potential to induce tumors in the tissues of contact and that this potential pertained largely and perhaps exclusively to the aromatic and saturate fractions. Coal tar, which had been shown to contain a greater

percentage of PAH's than asphalt [3], had a greater tumor-inducing potential, with 52-93% of the animals showing neoplastic formations under similar skin painting conditions [3,17,19]. The results from these skin-painting experiments support the hypothesis that the carcinogenic potencies of these mixtures increase with their polynuclear aromatic hydrocarbon contents.

Dose-response data for skin application of asphalt or asphalt in a solvent are not available in any individual report. However, it may be possible to obtain some rough information of this sort by comparing data from different experiments. In one experiment [3], an average total dose calculated to be 405 mg/animal and applied over 81 weeks caused neoplastic reactions in 6 (2.75%) of 218 mice. In another experiment [32], an average total dose calculated as 20-27 g/animal applied over 90 weeks caused neoplastic reactions in 2 (6.2%) of 32 mice. Because of the differences in asphalts used for each experiment, and because the neoplastic response has been shown to be dependent on details of the experimental procedure [45], comparison of dose-response relationships from different experiments is not feasible.

Injection of hot asphalt into experimental animals has also elicited neoplastic reactions. Subcutaneous injections of asphalt or asphalt mixed with olive oil resulted in a 2% incidence of neoplasms in 50 mice [32] and a 12.9% incidence of neoplasms in 62 mice [31]. Injection of the combined aromatic and saturate fractions of asphalt gave results that indicate a possible dose-response relationship [33]. Doses of 0.5 ml/animal produced neoplastic growths in 22.2% of 36 mice, while doses of 0.25 ml/animal, given 8 times for a total dose of 2 ml, produced neoplasms in 17.9% of 39

mice, and repeated doses of 1 ml/animal given for an approximate total dose of 9.7 ml produced neoplasms in 36.8% of 19 mice [33]. One problem with injecting different volumes of carcinogenic material is the possibility that the volumes administered play an important role in cancer induction [45]. The incidence of neoplastic growths following the injection of asphalt or asphalt in a carrier was definitely lower (1.5-12.9%) than the incidence (17.9-36.8%) of neoplasms after injection of the combined aromatic and saturate fractions of asphalt [19,31-33]. The differences in tumor incidence might be accounted for by the difference in the quantity of the PAH's applied to each group because of the concentration procedure for the saturate-aromatic fractions, and because of the higher doses administered. This indication is supported by an experiment in which the extracted volatiles from coal tar pitch, which are known to contain more PAH's than asphalt, were injected, with the subsequent development of neoplasms in 50 of 100 mice [19].

There have been few studies of the effects on animals of exposure to asphalt, and the authors' estimations of the potential hazards of exposure to asphalt are contradictory. Factors contributing to tumor induction, such as temperature of material applied, mechanical irritation of the skin from burns, tearing, and the application procedure, tumor-promoting effects, lipolysis, possible cocarcinogenicity of solvents and carriers used, and the influences of the total amount of material applied on the results from application of a constant dose of carcinogen, have been ignored for the most part. In light of the absence of reports suggesting an occupational carcinogenic hazard from asphalt or asphalt fumes, it appears that one or more of the aforementioned factors may be more

important in eliciting the neoplastic response than the asphalt itself applied.

Usual sanitation, work practices, and protective clothing may decrease the probability of expression of the carcinogenic potential that may be associated with asphalt fumes sufficiently for the probability of expression of this potential in workers with asphalt not to be statistically different from that of the carcinogenic potential from radiant energy of various characteristics and from incidental contaminants of the environment in the general population. No reports suggesting mutagenic or teratogenic effects of asphalt or asphalt fumes have been found in the literature. Properly designed epidemiologic studies to evaluate the potential risk of developing cancer from exposure to asphalt fumes have not been found.

Summary Tables of Exposure and Effect

The effects of exposures to asphalt fumes and asphalt presented in Chapter III are summarized in Tables XIII-3, XIII-4, and XIII-5. Data concerning exposure to asphalt fractions or to asphalt constituents are not included in these tables.

IV. ENVIRONMENTAL DATA

Environmental Concentrations

Determinations of worker exposure to asphalt fumes have not been found in the literature. However, several papers have reported analyses of the emissions from processes that use asphalt. The benzene-soluble fraction of the particulates and the benzo(a)pyrene levels in these particulates have been reported.

A 1975 study on emissions from asphalt hot-mix plants [5] listed a range of benzene-solubles of 0.3-2.8 mg/cu m for one plant and a range of 0.2-5.4 mg/cu m for another plant. The average benzo(a)pyrene concentrations in these fractions were 11 μ g/1,000 cu m and 19 μ g/1,000 cu m, respectively.

Thomas and Mukai [42] reported values of 2.9, 8.3, and 9.5 g/cu m for total particulates from heated asphalt kettles, but no measurable amount of benzo(a)pyrene was detected.

In 1974, Gerstle [43] analyzed the emissions from asphalt roofing manufacturing processes. These analyses showed that the polycyclic organic matter (polar aromatic compounds) constituted between 0.0003 and 0.0019% of the total particulate emissions and that BaP accounted for approximately 10% of this polycyclic organic matter.

Sampling and Analysis

There are currently no NIOSH validated sampling and analytical methods specific for asphalt fumes. Various methods have been used to

collect asphalt fumes in emission studies, including high-volume samplers using filtration [5] and impingers [42], but these studies have involved source performance standards and were not related to employee breathing-zone sampling.

Asphalt is almost completely soluble in benzene [6] and 99+% soluble in trichloroethylene [60]. Although a determination of the solubility of asphalt fumes has not been reported, it is reasonable to assume that they would have the same solubilities as the parent compounds since the fumes are not generated by excessive (cracking) temperatures. This indicates that, for the purpose of sampling and analysis, asphalt fumes are particulate polycyclic organic matter (PPOM), and that those methods developed by NIOSH and OSHA for the determination of PPOM in coal tar pitch volatiles can be used for the sampling and analysis of asphalt fumes.

More than 50 different types of instruments have been used for particulate sampling [61]. An appropriate sampler will allow the determination of either the mass concentration or the number concentration of the particles in a volume of gas. The instruments for measuring the mass concentration of a particulate can be classified into two categories according to whether or not they use preselectors, such as elutriators or preferably cyclones, to separate the coarser, nonrespirable particulates before collection. Because of the adhesive property of asphalt fumes, a preselector can induce error by trapping respirable particles. Collection without a preselector, however, can induce error by increasing the calculated mass of asphalt fumes by the amount of the total particulates that is actually in the nonrespirable size range.

Samples of PPOM have been collected on various filter media. In the 1960's, the method most often used was to draw air at a flowrate of 20-100 cu ft/min through a glass fiber filter with a high-volume sampler. A silver membrane filter for sampling coal tar pitch volatiles (or PPOM) was recommended by Richards et al [62] because it offered better weight stability than glass fiber, cellulose, or cellulose acetate filters. In a 1973 NIOSH evaluation of sampling and analysis for coke oven emissions, however, silver membrane filters were found to clog after a relatively short sampling time when emissions or moisture content was high [63]. This problem was eliminated when a glass fiber filter (without organic binder) was placed ahead of the silver filter within the sampling cassette. The combination of glass fiber and silver membrane filters, supported by a cellulose filter pad, then became the standard medium used by industrial hygienists for the collection of PPOM [64]. At present, the glass fiber filter without a silver membrane backup is considered adequate for measurement of PPOM.

After the samples are collected, the filters are extracted with an organic solvent. The most common method used for this extraction is the Soxhlet method with benzene as the solvent [64-66]. In the Soxhlet method, the filter used to collect the sample is weighed before and after exposure to the employee's environment and the difference in weight is a measure of the total particulate concentration [65]. The filter is removed from the holder and transferred to a Soxhlet apparatus, where the sample is extracted with hot benzene for 3 hours. After extraction, the filter is placed in a Millipore filtering apparatus, and the benzene extract is passed through the filters under suction to recover any particulates that

were dislodged during extraction. The filter is dried and reweighed, and any loss of weight is recorded as "benzene solubles" [64-66]. This benzene-extractable fraction has been used as an index of the presence of high-molecular-weight polycyclic hydrocarbons with carcinogenic properties [65]; however, this method is actually a nonspecific measurement of the organic material in the atmosphere [67].

In a critical analysis of the "benzene-soluble" extraction method, Seim et al [64] mentioned many conditions that caused major weight-loss errors in the results, including mechanical losses during handling and discontinuous films of fine particles on the glassware that cannot be recovered by washing with benzene. Other properties able to affect the measurement of PPOM were the boiling points of individual constituents, differences in solubility, reactivity to oxidants in the presence of light, stability on adsorbents, decomposition during solvent extraction, and collection temperatures and airflow rate [68].

The Soxhlet benzene-extraction method has been modified [69] to use ultrasonic extraction in benzene of the particulate matter on the filters. This ultrasonic procedure is faster (5 minutes) and easier to perform than the Soxhlet method. After the filtrate is extracted, the benzene solution is filtered through a glass fiber filter in a microfilter holder. The total material extracted is determined by weighing the residue from an aliquot of the extract that has been evaporated to dryness and making the necessary calculation.

Various methods have been developed for identifying and quantitating the constituents of asphalt fumes [70-72], but estimation of any one constituent is of little practical value in characterizing occupational

exposure because of the chemical variability of asphalts and their fumes [3-7]. Since the quantities of individual constituents of asphalt fumes are not a reliable indicator of the total concentration of fumes, NIOSH recommends that the concentration of total fumes be measured.

Total particulate concentrations should be monitored routinely by sampling with a preweighed glass-fiber filter as detailed in Appendix I [61,73]. Glass fiber filters have been selected because they will collect airborne particulates efficiently, are not hygroscopic, and do not clog as readily as membrane filters [61].

After collection of the sample, the weight of total particulates on the filter is determined by gravimetric analysis as detailed in Appendix II [61,73]. The final weight of the filter should be determined on the same balance that was used for determining the presampling weight. Before each weighing, the filter should be equilibrated in a constant humidity chamber, and a static charge neutralizer should be attached to the balance to improve the reproducibility of the weight determinations and thus enhance the gravimetric accuracy.

Because of the chemical variability of asphalt and asphalt fumes, there would be little utility or chemical consistency in attempting to estimate individual constituents of asphalt or asphalt fumes. Furthermore, there is probably an occupational hazard to laboratory personnel of undetermined magnitude associated with solvent extraction procedures involving benzene and possibly cyclohexane. A simple gravimetric method requiring no solvent extraction is therefore recommended for the estimation of occupational exposures to asphalt fumes, except for those high-dust situations in which nonextraction procedures might lead to erroneously high

estimates for asphalt fumes; in such cases, the solvent extraction procedure described in Appendix III is recommended.

Engineering Controls

Where the concentration of asphalt fumes exceeds the recommended occupational exposure limit, engineering controls must be instituted to decrease the concentration of asphalt fumes to the recommended limit or below. Industrial experience indicates that local-exhaust ventilation and closed-system operations are commonly used in control of asphalt fumes during the production of asphalt and the manufacture of products containing asphalt. Such systems must be used wherever possible to control the concentration of asphalt fumes at all permanent sites where asphalt is manufactured, processed, packaged, or used. Since the major uses of asphalt, paving and roofing, are carried on outdoors in temporary locations, closed-system operations are not always feasible. In these situations, care must be exercised to ensure that environmental conditions allow an adequate dispersal of the asphalt fumes so that the air concentration in an employee's breathing zone will not exceed the recommended occupational exposure limit. Closed-systems should operate under negative pressure when possible so that, if leaks develop, the airflow will be into the system. Closed-system operations are effective only when the integrity of the system is maintained; the system should therefore be inspected frequently, and leaks should be repaired promptly.

An exhaust-ventilation system may be used if a closed system is impractical. The principles that should be applied to control workplace air concentrations of asphalt fumes have been set forth in Industrial

Ventilation--A Manual of Recommended Practices [74], published by the American Conference of Governmental Industrial Hygienists; Fundamentals Governing the Design and Operation of Local Exhaust Systems, ANSI Z9.2-1971 [75], published by the American National Standards Institute; and NIOSH's Recommended Industrial Ventilation Guidelines [76]. These principles and practices have been used in the asphalt industry to control the environmental emissions and possible fire hazards so that asphalt processes can comply with air pollution abatement programs [21]. Ventilation systems require regular inspection and maintenance to ensure effective operation. Therefore, a program of routine scheduled inspections, including face velocity measurements of the collecting hood, inspection of the air mover and collector, and measurements of the workplace air concentrations of asphalt fumes, should be established. Any process changes that might affect the ventilation system or the operation being ventilated must be assessed prior to implementation to ensure that the engineering controls will continue to provide adequate protection for the employees; the effectiveness of engineering controls after the process change is completed should be checked as soon as possible after the change, and not later than 3 months.

V. WORK PRACTICES

Exposure to asphalt fumes at high concentrations has been shown to adversely affect the respiratory system [15, 19,27,30] and the eyes [29]. A continuing program of instruction on the proper use of respirators, including fitting, testing, and maintenance, and a procedure for assessing the employees' abilities to wear and work with respirators, eye protective devices, and other personal protective equipment should be developed and implemented by the employer. Respiratory and eye protective devices must be maintained in good working condition and must be routinely cleaned and inspected after each use. Respiratory, eye, and other protective equipment should not be used as a substitute for effective engineering controls. Because face shields and other devices for protecting the eyes may become obscured by impacted particles of asphalt, provisions should be made to clean the front surfaces of the shields or lenses.

Skin contact with asphalt has produced thermal burns [18] and dermatitis [27] in humans, as well as dermatitis and neoplastic growths in experimental animals [3,11,17,19,31,32]. Therefore, employees working with asphalt must be provided with and required to use protective clothing and equipment to reduce the possibility of adverse affects, regardless of the air concentration of asphalt fumes in the work atmosphere.

Gloves, aprons, and other personal protective devices must be kept clean and maintained in good condition to minimize the possibility of employee injury. Cotton gloves and work clothes and safety glasses with side shields should be adequate protection for employees potentially

exposed to hot asphalts.

Employees who may be exposed to hot asphalts while loading or unloading kettles or transports, or while sampling hot asphalts, should wear face shields (8-inch minimum) and heat-resistant, thermally insulated gloves. Employees working with cut-back or emulsified asphalts, which are fluid at room temperature (76 F; 25 C), should wear rubber gloves, rubber aprons, and goggles suitable for use around chemical hazards. All personal protective equipment should be cleaned frequently and respirator filter elements should be regularly inspected and replaced when necessary. Employers should ensure inspection and approval of all personal protective devices and should ensure that such equipment is stored in suitable, designated containers or locations at the job sites when they are not in use.

Work clothing should consist of a long-sleeved shirt fastened at the wrists and neck, long cuffless trousers, and heavy work shoes with nonmetallic safety toes. Clean work clothing should be put on before each work shift. At the end of the work shift, the employee should remove the soiled clothing and shower thoroughly before putting on street clothing. Soiled clothing should be drycleaned and then laundered before reuse. Handwashing and shower facilities with soap and separate lockers for work and street clothing should be provided by the employer at all permanent locations where exposure to asphalt or asphalt fumes is a possibility. At nonpermanent locations, employees should be provided with facilities for washing their hands and should be encouraged to shower after work.

Wide-mouth metal cans or pails without soldered joints should be used for sampling hot asphalt for quality control or other purposes [21].

Employees should use wire baskets or unbreakable pails to transport these samples.

A water supply for emergency first aid should be provided by a free-flowing hose at low pressure, an emergency shower, or other suitable water source. An adequate supply of a nonvolatile skin cleanser should be available. Eyewash fountains, bottles, or other suitable devices with fresh potable water, and emergency cold packs should also be available where eye or skin contact with asphalt is possible and should be inspected daily. Water in these containers should be changed daily. If eye contact with asphalt or serious skin burns should occur, the affected employee should see a physician as quickly as possible.

Smoking, drinking, eating, and dispensing or handling food should be prohibited in the immediate area of asphalt use or where asphalt fumes are present. The employer should designate at least one specific area for the activities listed above. Employees should be instructed to wash their hands before eating, drinking, or smoking, and before using toilet facilities.

All new or newly assigned asphalt workers should receive on-the-job training before they are allowed to work independently. Employers should determine that all employees are thoroughly familiar with all prescribed work practices before they are allowed to work alone. Employers should ensure that employees understand the instructions given to them. Periodic safety and health meetings should be conducted by the employer at least annually, and records of attendance should be maintained.

The duties of employees involved in maintenance and repair activities pose special problems of potential contact and exposure, especially during

work in enclosed systems or in operations involving ventilation system repair and maintenance. Written and oral instructions for recurring maintenance procedures, such as tank cleaning, production line changes, and repair or replacement of valves or conduits, should be provided to the appropriate personnel before they begin these or similar procedures.

Spills of hot asphalt may be dusted with washed sand or other nonhazardous mineral aggregates, such as vermiculite. Spills of asphalts that are fluid at room temperature should be dusted with a suitable oil absorbent. When the material can be handled safely, it should be removed from the work area and may be disposed of in a sanitary landfill according to local, state, and federal regulations. All permanent asphalt storage facilities should be appropriately diked to contain emergency spills.

Asphalt fumes can explode when they are concentrated in an enclosed environment and supplied with an ignition source [21]. Ventilation procedures should therefore be used where possible to minimize the concentration of fumes regardless of possible employee exposure, and possible ignition sources should be eliminated from areas in which fumes are likely to be concentrated. Areas and containers in which an explosive accumulation of asphalt fumes is possible should be posted and labeled with appropriate warnings of the explosive hazard.

The cut-back asphalts present the greatest fire and explosion hazard because of the volatile solvents used in their formulation. Because of their low flashpoints, they require extreme caution, and, whenever feasible, based on job requirements, they should always be handled and stored at temperatures below the flashpoint, not to exceed 212 F (100 C) [21]. Cut-back asphalts should not be heated or stored above the

flashpoint temperature. Because of the high heat retention of asphalts, contact with water or light hydrocarbons should be avoided to minimize the froth-over or boil-over hazards.

Written and oral instructions detailing proper handling procedures and work practices for loading and transporting bulk asphalt should be provided to each individual engaged in these activities by the producer or storage facility supplying the asphalt. Moreover, the employer should ensure that these individuals follow good work practices while loading, unloading, or transporting asphalt on his premises.

Entry into asphalt work areas associated with permanent facilities should be restricted to employees and guests accompanied by representatives of the employer [21]. The employer should be responsible for providing all guests with appropriate safety equipment and ensuring that it is used. A controlled-access system should be developed and used for all guests visiting the work areas.

When asphalt is used in the open, as in paving and roofing operations, employees should station themselves on the windward side of any source of asphalt emissions, as far as is possible. Sources of ignition, eg, smoking or open flames, should not be allowed within 15 meters (50 feet) of asphalt kettles, pavers, or, especially, spray rigs dispensing cut-back asphalt. Engines or burners on kettles should not be refueled while they are operating. No flammable liquid with a flashpoint of less than 38 C (100 F) should be used for any purpose within 15 meters (50 feet) of an operating asphalt kettle or any other source of ignition. All hoses and fittings should be checked daily for wear leading to leakage and should be repaired if leaks are discovered.

VI. DEVELOPMENT OF STANDARD

Basis for Previous Standards

The earliest workplace environmental limit for asphalt fumes was published in 1971 by the American Conference of Governmental Industrial Hygienists (ACGIH) [77], which recommended a threshold limit value (TLV) of 5 mg/cu m for asphalt fumes as a Time Weighted Average (TWA) concentration for an 8-hour workday in a 40-hour workweek. In the 1971 Documentation of the Threshold Limit Values for Substances in Workroom Air [78], this TLV was justified by the animal inhalation experiments of Hueper and Payne [19] and the health survey of petroleum asphalt workers conducted by Baylor and Weaver [27]. Supporting opinions were presented by the industrial hygienists who conducted the survey of working conditions for inclusion in the Documentation. They said that "...working conditions were satisfactory when asphalt fume levels were kept below 10 mg/cu m....Accordingly, a TLV of 5 mg/cu m is recommended to maintain good housekeeping conditions and reduce the risk of possible carcinogenicity [78]." In 1976, the ACGIH [79] proposed a tentative short-term exposure limit (STEL) of 10 mg/cu m for a 15-minute exposure to asphalt fumes. No change in the TLV was recommended in 1976 [79] or in the discussion of the basis for the TLV in the most recent (1974) Documentation [80].

In 1972, the Federal Register (37:24749, November 21, 1972) contained an interpretative rule of the term "coal tar pitch volatiles": "...coal tar pitch volatiles include the fused polycyclic hydrocarbons which volatilize from the distillation residues of coal, petroleum, wood, and other organic matter." This has been reprinted as 29 CFR 1910.1002. The

general philosophy behind this interpretation was that "all of these volatiles have the same basic composition and...present the same dangers to a person's health." The interpretation has been reinforced more recently in the Federal Register (41:46752, October 22, 1976) by the statement: "The existing standard will continue to apply to employee exposures to coal tar pitch volatiles outside of coke plants, such as the petroleum asphalt industry, including those parts of the steel plant other than the regulated area."

In 1973, objection to the original interpretative ruling was made by the American Petroleum Institute (API), on the basis that petroleum asphalt, unlike coal tar and coal tar pitch, is not a product of destructive (high-temperature) distillation and does not contain the comparatively high concentration of polycyclic compounds that is common in coal tar and coal tar pitch. The API proposed that the rule be amended by insertion of "destructive" before "distillation." This proposal was repeated by the Asphalt Institute in August of 1975. In September of 1975, the Division of Occupational and Radiological Health of the Tennessee Department of Public Health stated that it had found high concentrations of benzene-soluble materials in the carbon products industry, in which materials other than coal tar pitch that contain benzene-soluble volatiles are used. That department expressed the opinion that the coal tar pitch volatiles standard as stated was inapplicable to that industry and asked that an enforceable standard more specific for coal tar pitch volatiles be promulgated.

In October of 1975, a roofing manufacturer in New Jersey was cited by OSHA for allowing exposure of one of its employees to "coal tar pitch

volatiles associated with petroleum distillation residues" at a concentration in excess of the TWA concentration allowed in 29 CFR 1910.1000(a). This citation was vacated by a settlement agreement in January of 1977, the basis of the settlement being that exposures to benzene-soluble volatiles derived from petroleum asphalt at concentrations of up to 2.5 mg/cu m would be acceptable (personal communication, C. Lorr to E. Baier, August 1977). In essence, therefore, there appears to be a "standard" for exposure to volatiles from asphalt at 2.5 mg/cu m despite the interpretative rule of 29 CFR 1910.1002.

No standards for occupational exposure to asphalt fumes established by foreign countries have been found.

Basis for the Recommended Standard

(a) Permissible Exposure Limits

The literature on the biologic effects of exposure to asphalt fumes is often confusing and contradictory. Some of the problems arise from failure to distinguish between asphalt (residue from fractional distillation of petroleum) and tar (product from destructive distillation of petroleum, coal, peat, lignite, wood, or other organic substances) or pitch (residue from fractional distillation of tars). However, the toxicity of asphalt [3,17,19] and asphalt fumes [18,20] is substantially lower than that of coal tar, coal tar pitch, and their fumes. For further information on the toxicities of coal tar and coal tar pitch and their fumes, see NIOSH's Criteria for a Recommended Standard for Occupational Exposure to Coal Tar and Coal Tar Pitch. Because of this difference in toxicity, which has been documented in studies where both materials were

tested, care must be exercised in determining and defining exposure to asphalt fumes. Therefore, this recommended standard for asphalt fumes is based solely on their reported toxicity and not on their resemblance to fumes from tars and pitches.

Tumorigenic reactions have been observed in experimental animals after dermal application of undiluted asphalt [19,32] or asphalt mixed with solvents [3,17,19,31,32]. The incidence of these tumorigenic reactions, including both malignant and nonmalignant tumors, varies from zero to 9.5% for the hot asphalt and zero to 17.6% for the asphalt in a solvent. The mean incidence of tumor formation as calculated for the total number of animals beginning each experiment was 4.73% for the hot asphalt, 4.96% for the asphalt mixed with a solvent, and 4.69% for three control groups. Because of the lack of quantitative dose-response data for tumorigenic responses in these experiments, it is not possible to determine a safe limit for exposure to asphalt. The evidence from experiments with animals [19,32] indicates, however, that intimate contact must be maintained between the asphalt and the skin for a relatively long time before any neoplastic potential becomes manifest. The available data do not indicate a significant risk of carcinogenesis in human populations occupationally exposed to asphalt or asphalt fumes [27]. The usual personal hygiene of employees and the discontinuous nature of occupational exposure to asphalt and its fumes make a significant risk of carcinogenesis among such employees unlikely. In fact, only two human cases of carcinomas have been attributed to exposure to asphalt [16,24], but in one case [16] the presenters stated that asphalt may not have been the causative agent.

A health hazard survey [27] of 462 petroleum asphalt workers found no significant differences between the number and severity of current health problems in these workers and those in a control population of 379 refinery workers. In the past medical histories, however, the frequency of lung disease other than cancer in the asphalt workers was noted to be 1.96 times that in the control population (significant at $P < 0.05$, Chi Square). Whether these histories pertained to health problems prior to work with asphalt was not reported. Comparison of the frequency of lung disease in asphalt workers with data on lung disease in the general working population [28] showed no significant difference between the groups in past or present illness. Roofing workers reported suffering no adverse health effects when working with asphalt [18,20]. Four trucking companies with a total of more than 5,000 drivers exposed to asphalt fumes and dust reported no known cases of lung or skin disease attributable to asphalt, and three large roofing companies employing over 1,100 asphalt workers found no evidence of ill health related to asphalt exposure [27]. Thirty-one construction or paving companies reported one case of ill health attributed to asphalt in 11,478 man-years of work experience, and 15 state boards of health replying to a questionnaire mentioned only one case of nasal irritation and 14 cases of dermatitis attributed to asphalt or asphalt fumes among the cases of injury and ill health known to them [27]. Most of these data were obtained by questionnaire or by corroborative examinations of workers and were not accompanied by information relating to duration or concentration of exposures.

There also have been reports of respiratory effects in humans [15] and in animals [19,30] exposed to asphalt fumes. Quantitative

determinations of the concentrations of the fumes were not presented, but subjective descriptions of the environments as acrid and irritating to the nose and throat [15] or as containing dense oily fumes [30] indicated that concentrations were relatively high. Observed effects in humans have included burning sensations in the nose, throat, and chest, nasal mucous discharge, coughing, and expectoration, tonsillitis, pharyngitis, and loss of voice, bronchitis and increased vascularization of the bronchi, and emphysema and harsh respirations with rales [15]. It should be recalled, however, that the causative agent was not definitely identified as asphalt fumes. The following effects have been observed in animals after exposure to asphalt fumes at estimated concentrations of 40-2,000 mg/cu m for 2 years: pneumonitis with peribronchial adenomatosis, squamous cell metaplasia of the bronchial mucosa, bronchiectatic lumina, and emphysema [19,30].

In animal studies, pulmonary lesions were scattered [19,30]. The effects on both the upper and lower respiratory tracts were transient in humans exposed for less than 1 year [15]. The respiratory effects observed in animals after exposure to asphalt fumes were similar to those observed after inhalation exposure to nonspecific respiratory irritants at high concentrations, some animals being relatively refractory to the irritant. None of the reports in the literature demonstrates conclusively that asphalt fumes have a carcinogenic potential in humans or animals. Since so few cases of irritation in humans have been attributed to asphalt fumes even though the industrial experience with asphalt includes large numbers of workers over several decades, the occupational health hazard from these fumes appears to be minimal.

Adverse skin effects other than tumorigenesis from exposure to asphalt have also been observed in humans [18,27] and in animals [3,17,19,31,32]. The investigators have rarely reported dose or exposure concentrations, so that quantitative determination of a dose-response relationship is not possible. The adverse reactions reported in humans included occasional burns from hot asphalt [18] and 14 cases of dermatitis recorded by a state board of health [27] and attributed to exposure to asphalt. Adverse nontumorigenic skin effects reported in animals were similar to those that result from application of benzene or toluene, which were used as solvents for the asphalt. These effects included hair loss, chronic dermatitis, and dryness and scaling of the skin at the application site [3,17,19,31,32]. Asphalt fumes directed into the eyes of immobilized rabbits caused mild conjunctivitis and slight infiltration of the cornea, but both conditions disappeared after the exposure was discontinued [29].

The risk to the health of employees exposed to asphalt fumes is the result of the potential irritating actions of the fumes on the eyes and adjacent tissues and on the respiratory tract, and of the asphalt from which the fumes are generated on the skin or other tissues of contact. Although there have been few reports attributing adverse effects to occupational exposure to asphalt and asphalt fumes, animal data indicate a risk from exposure to these substances at high concentrations.

The particle sizes of asphalt fumes have been reported to be less than 5 μm [5], ie, in the respirable range. The data indicate that the effects of exposure to asphalt fumes at high concentrations are qualitatively and quantitatively similar to those of exposure to any nonspecific respiratory irritant, and while no experiment has clearly

demonstrated a direct carcinogenic hazard associated with asphalt fumes, NIOSH is concerned that future scientific investigations may indicate a greater occupational hazard from asphalt fumes than is currently documented in the literature. Concern for employee health requires that the possibility of short-term and long-term effects of asphalt and asphalt fumes be minimized. This concern has been precipitated by the paucity of credible toxicologic evidence and the confusion in the literature regarding asphalts, tars, and pitches. Inadequate scientific research in these particular areas has left unresolved issues, and these compel NIOSH to recommend a ceiling concentration limit of 5 mg/cu m based on total particulate. Limiting employee exposure to asphalt fumes to a concentration of not more than 5 mg/cu m should protect against possible respiratory or eye irritation over the entire normal working life of the individual and minimize the likelihood of a neoplastic response from exposure to asphalt fumes.

(b) Sampling and Analysis

Technology is currently available to sample and analyze asphalt fumes at the recommended occupational exposure limits and to provide necessary engineering controls. Sampling and analysis should be carried out by the recommended sampling and analytical methods for asphalt fumes as discussed in Chapter IV and presented in greater detail in Appendices I, II, and III. These methods provide for sampling of the workers' breathing zones by means of personal sampling pumps and glass fiber filters and for gravimetric determinations of particulates collected on the filters. Because of the physical properties of the particles of asphalt fumes, their deformability and adhesiveness, it is not practical to use a separation technique based

on particle size for separation of respirable from nonrespirable particulates when collecting breathing zone samples of asphalt fumes.

(c) Medical Surveillance and Recordkeeping

Respiratory irritation has been found in humans [15,27], and respiratory and eye irritation have been reported in animals [19,29,30] after exposure to asphalt fumes. Skin irritation and burns resulting from contact with hot asphalt have been reported in humans [18,27] and skin irritation and neoplastic reactions, both nonmalignant and malignant, have been produced in asphalt-exposed animals [3,17,19,31,32]. Although these reports are few and often contradictory, they do suggest a potential hazard from exposure to asphalt and asphalt fumes. A medical surveillance program should therefore be established to provide for the timely detection of any adverse health effects and should include preplacement and periodic medical examinations that give attention to the eyes, skin, respiratory system, respiratory function tests, and chest roentgenograms. Personnel potentially exposed to asphalt or asphalt fumes must be warned and advised of the possible adverse effects of exposure and must be informed of the signs and symptoms of the possible disorders. If eye contact with hot, cut-back, or emulsified asphalt occurs, the affected areas should be immediately flushed with water, and the employee should be examined by a physician. If substantial skin contact with hot asphalt occurs, the area should be immersed in cold water, dressed with an emergency cold pack, or packed with ice, and the employee should be examined by a physician as quickly as possible.

Because asphalt has the potential to produce skin neoplasms in experimental animals [11,31,32], all medical records should be kept for 30

years after termination of employment. This will allow time for future detection of delayed effects that may be related to the employee's known occupational exposure.

(d) Personal Protective Equipment and Clothing

Respiratory irritation has been reported in humans [15] and animals [19,30] exposed to high concentrations of asphalt fumes. Respirators should be available therefore and worn where concentrations of asphalt fumes exceed the recommended occupational exposure limit. The use of respirators should not be considered to be a substitute for proper engineering and administrative controls. Dermal and eye contact with liquid asphalt or asphalt fumes may induce irritation in humans [27] and in animals [3,17,19,29,31,32]. Care must therefore be exercised to ensure adequate protection against contact regardless of the air concentration of asphalt fumes. Personal protective equipment and clothing, including eye protective devices and gloves, should be available and should be worn where exposure to liquid asphalt is likely. Work practices that prevent skin and eye contact should be followed. A source of clean water and emergency cold packs should be available for immediate use if accidental contact occurs. Ice packs are useful if the skin comes into contact with molten asphalt.

(e) Informing Employees of Hazards

A continuing education program is an important part of a preventive hygiene program for employees occupationally exposed to such potentially hazardous materials as asphalt or asphalt fumes. Properly trained persons should periodically (at least annually) apprise employees of the possible sources of exposure to asphalt or asphalt fumes, the adverse effects associated with such exposures, the engineering controls and work practices

in use and being planned to limit such exposures, the first-aid measures to be taken upon accidental exposure, and the procedures used to monitor environmental controls and the health status of employees.

(f) Work Practices

Engineering controls must be used to maintain concentrations of asphalt fumes within the recommended environmental limits. During the time required to install adequate controls and equipment, to make process changes, to perform routine maintenance operations, or to make emergency repairs, exposure to asphalt fumes may be minimized by the use of respirators. However, respirators should not be used as a substitute for proper engineering controls in normal operations.

In the interest of good personal hygiene and work practices, it is recommended that food storage, handling, dispensing, and eating be prohibited in asphalt work areas, regardless of the concentrations of asphalt fumes. In addition, it is recommended that employees who work in an asphalt work area wash their hands thoroughly before eating, smoking, or using toilet facilities.

(g) Monitoring and Recordkeeping

Many employees come in contact with only small amounts or low concentrations of asphalt or asphalt fumes, usually only in the case of an accident, during maintenance procedures, or in open-air environments. Therefore, under normal working conditions, it should not be necessary to conduct extensive monitoring and surveillance procedures for employees with negligible contact. Where exposure to asphalt fumes at concentrations above one-half the recommended occupational exposure limit or to hot, cut-back, or emulsified asphalt can occur, adequate protection against the

potential irritating effects of asphalt and asphalt fumes must be ensured. Concern for employee health requires that protective measures be instituted at concentrations at or below the recommended workplace exposure limit to ensure that exposures stay below that limit. For this reason, an industrial hygiene survey should be conducted every 3 years in those occupational environments in which concentrations of asphalt fumes are at or below one-half the recommended occupational exposure limit. When concentrations are above one-half the recommended occupational exposure limit, annual personal monitoring is required. Records of monitoring and process changes should be retained for 30 years. This is in accord with the provisions of the Toxic Substances Control Act and will allow an analysis of the efficiency of engineering controls, of exposure potentials, and of the impact of process changes on the concentrations of the fumes and on potential exposure of employees.

VII. RESEARCH NEEDS

Proper assessment of the toxicity of asphalt fumes and evaluation of their potential hazard to the working population requires further animal and human studies. The following aspects of epidemiologic and toxicologic research are especially important.

Epidemiologic Studies

Detailed long-term epidemiologic studies, retrospective and prospective, of worker populations exposed to asphalt fumes are needed. Studies should involve both road workers and roofing workers. Care must be exercised to determine that these worker populations have not been exposed to fumes of tar or pitch or to dusts of asbestos or lime, because asphalt workers have frequently worked with materials containing these substances. Experimental studies have demonstrated that these substances are more toxic than asphalt fumes so that the assessments of the true hazard of asphalt fumes would be compromised by dual-exposure situations. As a minimum, epidemiologic studies should include environmental air measurements, medical and work histories, smoking and drinking histories, and body weight histories, pulmonary function studies, physical examinations with particular emphasis on respiratory, eye, and skin examinations, and comparisons with morbidity and mortality information from a carefully selected control population. Samples of urine collected from the cohort under study should be examined for mutagenic activity with tester strains of bacteria (Ames Tests). Additional studies should be performed to evaluate the phototoxic effects of asphalt or asphalt fumes.

Animal Studies

Adverse effects on the eyes and the respiratory tract of animals exposed to high concentrations of asphalt fumes [19,29,30] have been observed. These studies, however, did not include determinations of the concentrations of asphalt fumes to which the animals were exposed. Additional studies are necessary to determine the effects produced by exposure to asphalt fumes at known concentrations, especially in the range of the recommended occupational exposure limit. These studies should attempt to simulate the schedules of exposure in a normal working environment, so that their results could provide information relevant to workplace exposure conditions.

Studies of Carcinogenesis, Mutagenesis, Teratogenesis, and Effects on Reproduction

The available literature does not implicate asphalt fumes in carcinogenesis [19,30,27], although there are some indications that asphalt itself may be neoplastigenic in animal experiments in which it was applied to the skin [3,11,19,32] or injected subcutaneously [19,31-33,45]. The interplay of asphalt and other initiators and promoters of carcinogenesis should be investigated. Further research, including extensive long-term and multigeneration experiments, should be conducted to determine whether mutagenic, teratogenic, or other reproductive effects are caused by asphalt fumes. These experiments should be designed to simulate the exposure potential of a normal work situation.

Sampling and Analytic Studies

Studies are needed to improve the recommended sampling and analytical methods for asphalt fumes. These studies should be concentrated on techniques for separating the fumes from other airborne particulates. A comprehensive characterization of the chemical composition of the fumes generated by heating the asphalts derived from several crude oil sources is also needed, as is research to determine safe substitutes for benzene as an extraction solvent.

Biologic Monitoring

A simple, noninvasive method for detecting significant exposure to asphalt would be useful. A urine test that would signal such an exposure is desirable. Possibilities are detection of hydroxylated polycyclic hydrocarbons, of conjugated products with sulfuric or glucuronic acids or of increased excretions of sulfates, glucuronides, phenols, quinones, or mercapturic acids, in addition to the hydrocarbons themselves, in urine samples voided at the end of the workday or the workweek, depending on the frequency at which monitoring is desired. To be practical the monitoring method must be able to detect exposure during any 10-hour period, with 15-minute interruptions during both the morning and the afternoon periods and a 30-minute one at midday, to asphalt fumes at a concentration of not more than 2.5 mg/cu m, calculated on the basis of a 15-minute sampling period.

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IX. APPENDIX I

METHOD FOR SAMPLING ASPHALT FUMES IN AIR

The sampling method for airborne asphalt fumes is adapted from a general method for sampling airborne particulates [61,73] and is based on procedures developed by NIOSH and currently being used by OSHA.

General Requirements

Collect breathing zone samples that are representative of the individual employee's exposure. Collect enough samples to permit calculation of a TWA concentration for every operation or location in which there is exposure to asphalt fumes. At the time of sample collection, record the sampling location and conditions, equipment used, time and rate of sampling, name of the individual performing the sampling, and any other pertinent information.

Equipment for Air Sampling

(a) Filter: A 37-mm preweighed glass fiber filter, free of organic binders, mounted with backup pad in a two-piece polystyrene cassette.

(b) NIOSH-approved battery-operated personal sampling pump having a flowrate of at least 1 liter/minute.

(c) Calibration device: A calibration device such as is described in the Section on Calibration of Sampling Trains.

Calibration of Sampling Trains

Since the accuracy of an analysis can be no greater than the accuracy into which the volume of sample is measured, the accurate calibration of the sampling pump is essential. The frequency of calibration required depends on the use, care, and handling to which the pump is subjected. Pumps should be recalibrated if they have been misused or if they have just been repaired or received from a manufacturer. If the pump receives hard use, more frequent calibration may be necessary. Regardless of use, maintenance and calibration should be performed on a regular schedule, and records of these should be maintained.

Ordinarily, pumps should be calibrated in the laboratory both before and after they are used in the field and after they have been used to collect a large number of field samples. If extensive field sampling is performed, calibration may also be performed periodically during sampling to ensure the continuous satisfactory operation of the pump and sampler. The accuracy of calibration is dependent on the type of instrument used as a reference. The choice of calibration instrument will depend largely on where the calibration is to be performed. For laboratory calibration, a soapbubble meter is recommended (Figure XIII-2), although other standard calibrating instruments, such as a spirometer, Marriott's bottle, or dry-gas meter, can be used.

Instructions for calibration with the soapbubble meter follow. If another calibration device is selected, equivalent procedures should be

used. Since the flowrate is dependent on the pressure drop of the sampling device, the pump must be calibrated while being operated with a representative filter in the line. With a water manometer, the pressure drop should not exceed 13 inches of water.

(a) While the pump is running, check the voltage of the pump battery with a voltmeter to assure that it is adequate for calibration. Charge the battery if necessary.

(b) Turn on the pump and immerse the buret in the soap solution; draw bubbles up the inside until they are able to travel the entire length of the buret without bursting.

(c) Adjust the pump flow controller to provide the desired flowrate.

(d) Start a soapbubble up the buret and measure with a stopwatch the time the bubble takes to move from one calibration mark to another.

(e) Repeat the procedure in (d) at least three times, average the results, and calculate the flowrate by dividing the volume between the preselected marks by the time required for the soapbubble to traverse the distance.

(f) Data and records necessary for the calibration include volume measured, elapsed time, pressure drop, air temperature, atmospheric pressure, serial number of the pump, date, and the name of the person performing the calibration.

Air Sampling

(a) Collect personal samples as close to the employee's breathing zone as possible without interfering with the employee's work in order to

characterize the exposure for each job or specific operation. Sampling flowrates should be checked frequently. If filters become clogged to the extent that the selected airflow cannot be maintained, change the filters and initiate the collection of new samples.

(b) Collect samples using a NIOSH-approved portable sampling pump whose flowrate can be determined to an accuracy of $\pm 5\%$ at 1-3 liters/minute. Connect the pump to the cassette, which consists of a preweighed glass-fiber filter free of organic binders, mounted on a two-piece cassette holder and supported by a backup pad.

(c) Operate the pump at a known flowrate to sample a minimum volume of 90 liters. Record the total sampling time, and other data as in Section (f) above. Record the total sampling time. A sample size of 90 liters is recommended.

(d) With each batch of 10 samples, submit one filter from the same lot used for sample collection and label it as a blank. Subject it to exactly the same handling as the samples, but do not draw any air through it.

X. APPENDIX II

ANALYTICAL METHOD (TOTAL PARTICULATE) FOR ASPHALT FUMES

A gravimetric analysis for total particulates should be performed with a preweighed glass-fiber filter [61,73].

Principle of the Method

Air samples are drawn through glass-fiber filters; the filters are then analyzed by a general gravimetric particulate method.

Range and Sensitivity

The range and sensitivity are based on the capabilities of the weighing instrument and the relationship of the particulate weight to the tare weight of the filter.

Interferences

Other particulates in the workplace air will also be collected by the filter and will cause a high reading.

Precision and Accuracy

The precision and accuracy of the gravimetric sampling method is defined by the limit of sensitivity of the balance used to weigh the filter and the interference present in the volume of sampled air.

Apparatus

- (a) Balance reading to 0.005 mg.
- (b) Desiccator or similar controlled humidity chamber.

Analysis of Samples

(a) A glass-fiber filter is placed in a chamber over an aqueous sulfuric acid solution for 24 hours to bring the filter to a constant weight at 50% humidity.

(b) The weight of the glass-fiber filter is recorded to the nearest 0.01 mg. A nuclear static eliminator on the balance will remove static charges that might interfere with obtaining accurate, reproducible weights of the filter.

(c) A known volume of air is drawn through the preweighed glass-fiber filter to collect airborne dust, including airborne asphalt fumes.

(d) After sampling, the filter is replaced in the chamber for 24 hours and again brought to a constant weight at 50% humidity.

(e) The filter is weighed on the balance used for the preweighing, and the weight is recorded to the nearest 0.01 mg. The difference in the initial and final weights of the filter, divided by the known volume of air sampled, equals the environmental concentration for asphalt fumes as total particulates.

Calculations

The concentration of asphalt fumes in air is expressed as the weight of the total particulates in mg/cu m of air sampled (mg/cu m).

- (a) Weight of particulates:

$$W_p = W_f - W_i$$

where:

W_p = weight of particulates in mg
 W_f = post-sampling weight of filter in mg
 W_i = pre-sampling weight of filter in mg

- (b) Volume of air sampled:

$$V_s = \frac{V \times P \times 298 \times 1,000}{760(T+273)}$$

where:

V_s = volume of air in cu m at standard conditions
 V = volume of air sampled in liters
 P = barometric pressure in mm Hg
 T = temperature of sample air, C

XI. APPENDIX III

ANALYTICAL METHOD (SOLVENT EXTRACTION) FOR ASPHALT FUMES

A solvent extraction method should be used for analysis of asphalt fumes collected in dusty atmospheres.

Principle of the Method

The cyclohexane-soluble material in the particulates on the glass fiber filters is extracted ultrasonically. Blank filters are extracted along with, and in the same manner as, the samples. After extraction, the cyclohexane solution is filtered through a fritted glass funnel. The total material extracted is determined by weighing a dried aliquot of the extract.

Range and Sensitivity

When the electrobalance is set at 1 mg, this method can detect 75-2,000 $\mu\text{g}/\text{sample}$.

Precision and Accuracy

When nine aliquots of a benzene solution from a sample of aluminum-reduction plant emissions containing 1,350 $\mu\text{g}/\text{sample}$ were analyzed, the standard deviation was 25 μg [73]. Experimental verification of this method using cyclohexane is not yet complete.

Advantages and Disadvantages of the Method

(a) Advantages

This procedure is much faster and easier to perform than the Soxhlet method.

(b) Disadvantages

If the whole sample is not used for cyclohexane-extraction analysis, small weighing errors make large errors in final results.

Apparatus

- (a) Ultrasonic bath, 90 Kc, 60 watts, partially filled with water.
- (b) Ultrasonic generator, Series 200, 90 Kc, 60 watts.
- (c) Electrobalance capable of weighing to 1 μ g.
- (d) Stoppered glass test tube, 150- x 16-mm.
- (e) Teflon weighing cups, 2-ml, approximate tare weight 60 mg.
- (f) Dispensing bottle, 5-ml.
- (g) Pipets, with 0.5-ml graduations.
- (h) Glass fiber filters, 37-mm diameter, Gelman Type A or equivalent.
- (i) Silver membrane filters, 37-mm diameter, 0.8- μ m pore size.
- (j) Vacuum oven.
- (k) Tweezers.
- (l) Beaker, 50-ml.
- (m) Glassine paper, 3.5- x 4.5-inches.
- (n) Wood application sticks for manipulating filters.
- (o) Funnels, glass-fritted, 15-ml.

- (p) Graduated evaporative concentrator, 10-ml.

Reagents

- (a) Cyclohexane, ACS nanograde reagent.
(b) Dichromic acid cleaning solution.
(c) Acetone, ACS reagent grade.

Procedure

(a) All extraction glassware is cleaned with dichromic acid cleaning solution, rinsed first with tap water, then with deionized water followed by acetone, and allowed to dry completely. The glassware is rinsed with nanograde cyclohexane before use. The Teflon cups are cleaned with cyclohexane, then with acetone.

(b) Pre-weigh the Teflon cups to one hundredth of a milligram (0.01 mg).

(c) Remove top of cassette and hold over glassine paper. Remove plug on bottom of cassette. Insert end of application stick through hole and gently raise filters to one side. Use tweezers to remove filters, and loosely roll filters around tweezers. Slide rolled filters into test tube and push them to bottom of tube with application stick. Add any particulates remaining in cassette and on glassine paper to test tube.

(d) Pipet 5 ml of cyclohexane into test tube from dispensing bottle.

(e) Put test tube into sonic bath so that water level in bath is above liquid level in test tube. Do not hold tube in hand while sonifying.

A 50-ml beaker filled with water to level of cyclohexane in tube works very well.

- (f) Sonify sample for 5 minutes.
- (g) Filter the extract in 15-ml medium glass-fritted funnels.
- (h) Rinse test tube and filters with two 1.5-ml aliquots of cyclohexane and filter through the fritted-glass funnel.
- (i) Collect the extract and two rinses in the 10-ml graduated evaporative concentrator.
- (j) Evaporate down to 1 ml while rinsing the sides with cyclohexane.
- (k) Pipet 0.5 ml of the extract to preweighed Teflon weighing cup. These cups can be reused after washing with acetone.
- (l) Evaporate to dryness in a vacuum oven at 40 C for 3 hours.
- (m) Weigh the Teflon cup. Use counterweighing techniques on electrobalance with full scale range of 1 mg to determine weight of aliquot to nearest microgram. The weight gain is due to the cyclohexane-soluble residue.

Calculations

The amount of cyclohexane-extractable fraction present in the sample (in mg) may be determined according to the following equation:

$$\text{mg/sample} = 2 \times (\text{wt sample aliquot (mg)} - \text{wt blank aliquot (mg)})$$

The amount of cyclohexane-extractable fraction present in the air may then be determined according to the following equation:

$$\text{mg/cu m} = \frac{\text{mg/sample}}{\text{air volume collected (cu m)}}$$

XII. APPENDIX IV
MATERIAL SAFETY DATA SHEET

The following items of information which are applicable to a specific product or material shall be provided in the appropriate block of the Material Safety Data Sheet (MSDS).

The product designation is inserted in the block in the upper left corner of the first page to facilitate filing and retrieval. Print in upper case letters as large as possible. It should be printed to read upright with the sheet turned sideways. The product designation is that name or code designation which appears on the label, or by which the product is sold or known by employees. The relative numerical hazard ratings and key statements are those determined by the rules in Chapter V, Part B, of the NIOSH publication, An Identification System for Occupationally Hazardous Materials. The company identification may be printed in the upper right corner if desired.

(a) Section I. Product Identification

The manufacturer's name, address, and regular and emergency telephone numbers (including area code) are inserted in the appropriate blocks of Section I. The company listed should be a source of detailed backup information on the hazards of the material(s) covered by the MSDS. The listing of suppliers or wholesale distributors is discouraged. The trade name should be the product designation or common name associated with the material. The synonyms are those commonly used for the product, especially formal chemical nomenclature. Every known chemical designation or

competitor's trade name need not be listed.

(b) Section II. Hazardous Ingredients

The "materials" listed in Section II shall be those substances which are part of the hazardous product covered by the MSDS and individually meet any of the criteria defining a hazardous material. Thus, one component of a multicomponent product might be listed because of its toxicity, another component because of its flammability, while a third component could be included both for its toxicity and its reactivity. Note that a MSDS for a single component product must have the name of the material repeated in this section to avoid giving the impression that there are no hazardous ingredients.

Chemical substances should be listed according to their complete name derived from a recognized system of nomenclature. Where possible, avoid using common names and general class names such as "aromatic amine," "safety solvent," or "aliphatic hydrocarbon" when the specific name is known.

The "%" may be the approximate percentage by weight or volume (indicate basis) which each hazardous ingredient of the mixture bears to the whole mixture. This may be indicated as a range or maximum amount, ie, "10-40% vol" or "10% max wt" to avoid disclosure of trade secrets.

Toxic hazard data shall be stated in terms of concentration, mode of exposure or test, and animal used, eg, "100 ppm LC50-rat," "25 mg/kg LD50-skin-rabbit," "75 ppm LC man," or "permissible exposure from 29 CFR 1910.1000," or, if not available, from other sources of publications such as the American Conference of Governmental Industrial Hygienists or the American National Standards Institute Inc. Flashpoint, shock sensitivity

or similar descriptive data may be used to indicate flammability, reactivity, or similar hazardous properties of the material.

(c) Section III. Physical Data

The data in Section III should be for the total mixture and should include the boiling point and melting point in degrees Fahrenheit (Celsius in parentheses); vapor pressure, in conventional millimeters of mercury (mmHg); vapor density of gas or vapor (air = 1); solubility in water, in parts/hundred parts of water by weight; specific gravity (water = 1); percent volatiles (indicated if by weight or volume) at 70 degrees Fahrenheit (21.1 degrees Celsius); evaporation rate for liquids or sublimable solids, relative to butyl acetate; and appearance and odor. These data are useful for the control of toxic substances. Boiling point, vapor density, percent volatiles, vapor pressure, and evaporation are useful for designing proper ventilation equipment. This information is also useful for design and deployment of adequate fire and spill containment equipment. The appearance and odor may facilitate identification of substances stored in improperly marked containers, or when spilled.

(d) Section IV. Fire and Explosion Data

Section IV should contain complete fire and explosion data for the product, including flashpoint and autoignition temperature in degrees Fahrenheit (Celsius in parentheses); flammable limits, in percent by volume in air; suitable extinguishing media or materials; special firefighting procedures; and unusual fire and explosion hazard information. If the product presents no fire hazard, insert "NO FIRE HAZARD" on the line labeled "Extinguishing Media."

(e) Section V. Health Hazard Information

The "Health Hazard Data" should be a combined estimate of the hazard of the total product. This can be expressed as a TWA concentration, as a permissible exposure, or by some other indication of an acceptable standard. Other data are acceptable, such as lowest LD50 if multiple components are involved.

Under "Routes of Exposure," comments in each category should reflect the potential hazard from absorption by the route in question. Comments should indicate the severity of the effect and the basis for the statement if possible. The basis might be animal studies, analogy with similar products, or human experiences. Comments such as "yes" or "possible" are not helpful. Typical comments might be:

Skin Contact--single short contact, no adverse effects likely; prolonged or repeated contact, possibly mild irritation.

Eye Contact--some pain and mild transient irritation; no corneal scarring.

"Emergency and First Aid Procedures" should be written in lay language and should primarily represent first-aid treatment that could be provided by paramedical personnel or individuals trained in first aid.

Information in the "Notes to Physician" section should include any special medical information which would be of assistance to an attending physician including required or recommended preplacement and periodic medical examinations, diagnostic procedures, and medical management of overexposed employees.

(f) Section VI. Reactivity Data

The comments in Section VI relate to safe storage and handling of hazardous, unstable substances. It is particularly important to highlight instability or incompatibility to common substances or circumstances, such as water, direct sunlight, steel or copper piping, acids, alkalies, etc. "Hazardous Decomposition Products" shall include those products released under fire conditions. It must also include dangerous products produced by aging, such as peroxides in the case of some ethers. Where applicable, shelf life should also be indicated.

(g) Section VII. Spill or Leak Procedures

Detailed procedures for cleanup and disposal should be listed with emphasis on precautions to be taken to protect employees assigned to cleanup detail. Specific neutralizing chemicals or procedures should be described in detail. Disposal methods should be explicit including proper labeling of containers holding residues and ultimate disposal methods such as "sanitary landfill," or "incineration." Warnings such as "comply with local, state, and federal antipollution ordinances" are proper but not sufficient. Specific procedures shall be identified.

(h) Section VIII. Special Protection Information

Section VIII requires specific information. Statements such as "Yes," "No," or "If necessary" are not informative. Ventilation requirements should be specific as to type and preferred methods. Respirators shall be specified as to type and NIOSH or US Bureau of Mines approval class, ie, "Supplied air," "Organic vapor canister," etc. Protective equipment must be specified as to type and materials of construction.

(i) Section IX. Special Precautions

"Precautionary Statements" shall consist of the label statements selected for use on the container or placard. Additional information on any aspect of safety or health not covered in other sections should be inserted in Section IX. The lower block can contain references to published guides or in-house procedures for handling and storage. Department of Transportation markings and classifications and other freight, handling, or storage requirements and environmental controls can be noted.

(j) Signature and Filing

Finally, the name and address of the responsible person who completed the MSDS and the date of completion are entered. This will facilitate correction of errors and identify a source of additional information.

The MSDS shall be filed in a location readily accessible to employees exposed to the hazardous substance. The MSDS can be used as a training aid and basis for discussion during safety meetings and training of new employees. It should assist management by directing attention to the need for specific control engineering, work practices, and protective measures to ensure safe handling and use of the material. It will aid the safety and health staff in planning a safe and healthful work environment and in suggesting appropriate emergency procedures and sources of help in the event of harmful exposure of employees.

MATERIAL SAFETY DATA SHEET

I PRODUCT IDENTIFICATION		
MANUFACTURER'S NAME		REGULAR TELEPHONE NO EMERGENCY TELEPHONE NO
ADDRESS		
TRADE NAME		
SYNONYMS		
II HAZARDOUS INGREDIENTS		
MATERIAL OR COMPONENT	%	HAZARD DATA
III PHYSICAL DATA		
BOILING POINT, 760 MM HG		MELTING POINT
SPECIFIC GRAVITY (H ₂ O = 1)		VAPOR PRESSURE
VAPOR DENSITY (AIR = 1)		SOLUBILITY IN H ₂ O, % BY WT
% VOLATILES BY VOL		EVAPORATION RATE (BUTYL ACETATE = 1)
APPEARANCE AND ODOR		

IV FIRE AND EXPLOSION DATA					
FLASH POINT (TEST METHOD)				AUTOIGNITION TEMPERATURE	
FLAMMABLE LIMITS IN AIR, % BY VOL.			LOWER		UPPER
EXTINGUISHING MEDIA					
SPECIAL FIRE FIGHTING PROCEDURES					
UNUSUAL FIRE AND EXPLOSION HAZARD					
V HEALTH HAZARD INFORMATION					
HEALTH HAZARD DATA					
ROUTES OF EXPOSURE					
INHALATION					
SKIN CONTACT					
SKIN ABSORPTION					
EYE CONTACT					
INGESTION					
EFFECTS OF OVEREXPOSURE					
ACUTE OVEREXPOSURE					
CHRONIC OVEREXPOSURE					
EMERGENCY AND FIRST AID PROCEDURES					
EYES					
SKIN:					
INHALATION:					
INGESTION					
NOTES TO PHYSICIAN					

VI REACTIVITY DATA
CONDITIONS CONTRIBUTING TO INSTABILITY
INCOMPATIBILITY
HAZARDOUS DECOMPOSITION PRODUCTS
CONDITIONS CONTRIBUTING TO HAZARDOUS POLYMERIZATION
VII SPILL OR LEAK PROCEDURES
STEPS TO BE TAKEN IF MATERIAL IS RELEASED OR SPILLED
NEUTRALIZING CHEMICALS
WASTE DISPOSAL METHOD
VIII SPECIAL PROTECTION INFORMATION
VENTILATION REQUIREMENTS
SPECIFIC PERSONAL PROTECTIVE EQUIPMENT
RESPIRATORY (SPECIFY IN DETAIL)
EYE
GLOVES
OTHER CLOTHING AND EQUIPMENT

IX SPECIAL PRECAUTIONS

PRECAUTIONARY
STATEMENTS

OTHER HANDLING AND
STORAGE REQUIREMENTS

PREPARED BY _____

ADDRESS _____

DATE _____

XIII. TABLES AND FIGURES

TABLE XIII-1

CONCENTRATION RANGE FOR ASPHALT HOT-MIX EMISSIONS

Compound	New Jersey Range	North Carolina Range
	<u>ppm/vol</u>	
Methane	2 - 3	2 - 3
C2-C6 hydrocarbons	<1**	<1**
Hydrogen sulfide	<0.2*- 1.5	<0.2*
Carbonyl sulfide	<0.2*	<0.2*
Mercaptans	<0.2*	<0.2*
Sulfur dioxide	<2*	<0.5*
Carbon monoxide	4 - 6	3
Aldehydes	<0.1*	0.3 -0.4
Phenols	<1	<1**
Ozone	<0.1	-
Nitrogen dioxide	<0.1	0.05-0.08
	<u>mg/cu m</u>	
C7-C14 hydrocarbons	3 - 9	3 -5
High volume sample		
Total particulates	2.6 - 6.4	0.5 -5.7
Benzene soluble	0.3 - 0.5	0.2 -5.4
	<u>µg/1,000 cu m</u>	
Pyrene	44 - 240, 107 avg	96
Benz(a)anthracene	5 - 24, 11 "	32 - 38
Benzo(a)pyrene	3 - 20, 11 "	14 - 22
Benzo(e)pyrene	14 - 40, 26 "	Not found
Perylene	5 - 16, 12 "	6, 6

TABLE XIII-1 (CONTINUED)

CONCENTRATION RANGE FOR ASPHALT HOT-MIX EMISSIONS

Compound	New Jersey Range	North Carolina Range
	<u>µg/cu m</u>	
Nickel	0.005	0.04
Vanadium	0.02-0.08	<0.1*
Cadmium		<0.05*
Lead		<0.05*

*Not detected. If present at all, the compound was at a concentration below that as shown. The less than (<) value represents the sensitivity of the sampling and analysis procedure.

**Small amount detected; less than value shown

Adapted from reference 5

TABLE XIII-2

SOME USES AND APPLICATIONS OF ASPHALT

AGRICULTURE

Cattle sprays
 Dampproofing and waterproofing
 buildings, structures
 Disinfectants
 Fencepost coating
 Mulches
 Mulching paper
 Paved barn floors, barnyards, feed
 platforms, etc
 Protecting tanks, vats, etc
 Protection for concrete structures
 Tree paints
 Water and moisture barriers
 Wind and water erosion control
 Weather modification areas

Walls, siding, ceilings
 Acoustical blocks, compositions,
 felts
 Architectural decoration
 Asbestos cement panels, felt
 Bricks
 Brick siding
 Building blocks, papers
 Dampproofing coatings, compositions
 Insulating board, fabrics, felts,
 paper
 Joint filler compounds
 Masonry coatings
 Plaster boards
 Putty, asphalt
 Siding compositions
 Soundproofing
 Stucco base
 Wallboard

BUILDINGS

Floors
 Dampproofing and waterproofing
 Floor compositions, tiles, covering
 Insulating fabrics, papers
 Step treads

Miscellaneous
 Air-drying paints, varnishes
 Artificial lumber
 Ebonized lumber
 Insulating paints
 Plumbing, pipes
 Treated awnings

Roofing

Asbestos felt
 Building papers
 Built-up roof adhesives, felts,
 primes
 Caulking compounds
 Cement waterproofing compounds
 Cleats for roofing
 Glass wool compositions
 Insulating fabrics, felts, papers
 Joint filler compounds
 Laminated roofing, shingles
 Liquid roof coatings
 Plastic cements
 Shingles

HYDRAULICS and EROSION CONTROL

Canal linings, sealants
 Catchment areas, basins
 Dam groutings
 Dam linings, protection
 Dike protection
 Ditch linings
 Drainage gutters, structures
 Embankment protection
 Groins
 Jetties
 Levee protection
 Mattresses for levee and bank
 protection
 Membrane linings, waterproofing

TABLE XIII-2 (CONTINUED)

SOME USES AND APPLICATIONS OF ASPHALT

Reservoir linings	Compositions
Revetments	Black grease
Sand dune stabilization	Buffing compounds
Sewage lagoons, oxidation ponds	Cable splicing compound
Swimming pools	Embalming
Waste ponds	Etching compositions
Water barriers	Extenders, rubber, other
	Explosives
	Fire extinguisher compounds
	Joint fillers
	Lap cement
	Lubricating grease
	Pipe coatings, dips, joint seals
	Plastic cements
	Plasticizers
	Preservatives
	Printing inks
	Well-drilling fluid
	Wooden cask liners
<u>INDUSTRIAL</u>	
Aluminum foil compositions using asphalt	
Backed felts	
Conduit insulation, lamination	
Insulating boards	
Paint compositions	
Papers	
Pipe wrapping	
Roofing, shingles	
Automotive	Impregnated, treated materials
Acoustical compositions, felts	Armoured bituminized fabrics
Brake linings	Asbestos compositions
Clutch facings	Burlap impregnation
Floor sound deadeners	Canvas treating
Friction elements	Carpeting medium
Insulating felts	Deck cloth impregnation
Panel boards	Fabrics, felts
Shim strips	Mildew prevention
Tacking strips	Packing papers
Underseal	Pipes and pipe wrapping
	Planks
	Rugs, asphalt base
	Sawdust, cork, asphalt, composition
	Textiles, waterproofing
	Tiles
	Treated leather
	wrapping papers
Electrical	
Armature carbons, windings	Paints, varnishes, etc
Battery boxes, carbons	Acidproof enamels, mastics, varnishes
Electrical insulating compounds, papers, tapes, and coatings	Acid resistant coatings
Junction box compound	Air-drying paints, varnishes
Molded conduits	

TABLE XIII-2 (CONTINUED)

SOME USES AND APPLICATIONS OF ASPHALT

Anticorrosive and antifouling paints	Highways, roads, streets, shoulders
Antioxidants and solvents	Parking lots, driveways
Base for solvent compositions	PCC underseal
Baking and heat resistant enamals	Roof-deck parking
Boat deck sealing compound	Sidewalk, footpaths
Lacquers, japans	Soil stabilization
Marine enamels	
Miscellaneous	<u>RAILROADS</u>
Belting	Ballast-treatment
Blasting fuses	Curve lubricant
Briquet binders	Dust laying
Burial vaults	Paved ballast, subballast
Cashing molds	Paved crossings, freight yards, station platforms
Clay articles	Rail fillers
Clay pigeons	Railroad ties
Depilatory	Tie impregnating, stabilization
Expansion joints	
Flowerpots	<u>RECREATION</u>
Foundry cores	Paved surfaces for:
Friction tape	Dance pavilions
Fuel	Drive-in movies
Gaskets	Gymnasiums, sports arenas
Imitation leather	Playgrounds, school yards
Mirror backing	Race tracks
Phonograph records	Running tracks
Rubber, molded compositions	Skating rinks
Shoe fillers, soles	Swimming and wading pools
Table tops	Tennis courts, handball courts
<u>PAVING</u>	
Airport runways, taxiways, aprons, etc	
Asphalt blocks	
Brick fillers	
Bridge deck surfacing	
Crack fillers	
Curbs, gutters, drainage ditches	
Floors for buildings, warehouses, garages, etc	

Adapted from reference 8

TABLE XIII-3

EFFECTS OF OCCUPATIONAL EXPOSURE TO
ASPHALT OR ASPHALT FUMES

Number Exposed	Length of Exposure	Effects	Reference
22*	Several mon	Coughing with expectoration, burning sensation in throat and chest, frequent hoarseness, headache, nasal mucous discharge	15
22*	Several yr	Coughing with expectoration, nasal mucous discharge and inflammation, changes in vocal timbre, frequent loss of voice, tonsillitis, pharyngitis, acute febrile bronchitis, nosebleeds; 10 cases of rhinitis, 13 cases of oropharyngitis, 4 cases of laryngitis, 19 cases of bronchitis; harsh respirations with rales, basal hypophonesis with tympanic zones from emphysema, increased vascularization in bronchial areas	15
34	-	Occasional thermal burns	18
17	-	Virtually no irritation from asphalt	20
1*	31 yr	Blackish specks in sputum, chronic bronchitis, skin rashes, complete loss of voice, squamous cell carcinoma of the left vocal cord	16
462	5 yr or more	Skin cancer in 2, stomach cancer in 1; lung disease in 40, primarily bronchitis with some cases of asthma and some of emphysema; skin disease, mainly dermatitis, in 26; hypertension in 27; peptic ulcer in 12; heart disease in 17; effects not statistically different from 379 control workers	27
-**	11,478 man-yr	Ill health in 1 with no details	
1,000+	-	No evidence of ill health	27

TABLE XIII-3 (CONTINUED)

EFFECTS OF OCCUPATIONAL EXPOSURE TO
ASPHALT OR ASPHALT FUMES

Number Exposed	Length of Exposure	Effects	Reference
112	12+ yr avg	No evidence of ill health	27
-***	-	Headache in 1, silicosis in 1, leukoplakia in 1, dermatitis in 1	27
-****	-	Nasal irritation in 1, dermatitis in 14	

*Causative agent probably not asphalt

**Data obtained from 31 construction or paving companies

***Data obtained from 6 large insurance companies

****Data obtained from 15 State Boards of Health or State Highway Commissions

TABLE XIII-4

EFFECTS OF EXPOSURE TO ASPHALT FUMES ON ANIMALS

Species and Number	Exposure Concentration and Duration	Effects	Reference
Rabbits -	Dense, oily fumes	Minor, transient conjunctivitis, slight infiltration of the cornea; no controls	29
Guinea pigs (30)	0.4 - 2 g/hr*	Extensive, chronic fibrosing pneumonitis, peribronchial adenomatosis, squamous cell metaplasia of the bronchial mucosa, bronchiectatic lumina; no controls	19
Rats (65)	5 hr/d 4 d/wk for 2 yr		
Mice (30)	0.74- 0.93 g/hr* 6 - 7.5 hr/d 5 d/wk for a maximum of 401 exposures	Bronchitis, pneumonitis, abscess formation, loss of cilia, epithelial atrophy and fragmentation, necrosis, flattening of the epithelium, 1 bronchial adenoma, occasional epithelial hyperplasia and emphysema with focal lung collapse, peribronchial round-cell infiltration, and bronchial dilatation; no controls	30

*Asphalt lost from heated container by volatilization or decomposition

TABLE XIII-5

SUMMARY OF EFFECTS OF EXPOSURE TO ASPHALT ON ANIMALS

Species and Number	Route	Exposure		Effects	Reference
		Amount, Strength, and Diluent	Frequency and Duration		
Mice (32)	Dermal	75-100 mg - -	1- 3/wk 22-270 paintings	Chronic dermatitis; lung adenoma, 1; papilloma at painting site, 1; tumor of skin accessory structure origin, 1; no controls	32
Mice (40)	"	75-100 mg - -	- 16-240 paintings	Chronic dermatitis; epidermoid carcinomas, 3; papillomas, 2; no controls	32
Mice (20)	"	20- 30 mg 90% Toluene	3/wk 284 paintings	Chronic dermatitis; epidermoid carcinomas, 9; lung adenomas, 2; hair loss, scaling of skin, 2 papilloma in 15 controls	32
Mice (218)	"	2.5 mg - Benzene	2/wk 81 wk	Hyperplasia of epidermis, inflammation, ulceration, and abcess formation, amyloid accumulation in spleen and kidneys; 6 skin tumors, including 1 carcinoma; similar signs in controls, including 1 nonmalignant papilloma, no carcinomas	3
Mice (50) Rabbits (6)	"	- - -	2/wk 2 yr max	No neoplasms	19
Mice (250)	"	- - Acetone	"	Skin carcinomas, 2; papillomas, 2; leukemias, 4; Kupffer-cell sarcoma, 1	19

TABLE XIII-5 (CONTINUED)

SUMMARY OF EFFECTS OF EXPOSURE TO ASPHALT ON ANIMALS

Species and Number	Route	Exposure		Effects	Ref- erence
		Amount, Strength, and Diluent	Frequency and Duration		
Mice (68)	Dermal	- - Benzene	2/wk -	Epidermoid carcinomas, 12; papillomas, 5; hair loss at painting site, dry, scaling skin in test animals and in 63 controls	31
Mice (50)	"	- 40% Benzene	1/wk 19 mon	Squamous cell carcinoma, 1; sebaceous adenoma, 1; pul- monary adenomas and adeno- carcinomas in 5 mice; epi- dermal atrophy, focal hyper- plasia, hyperkeratosis, acute and chronic inflamma- tion; epidermal atrophy, focal hyperplasia, hair follicle atrophy in controls	17
"	"	- 40% Benzene	"	Subcutaneous fibrosarcoma, 1; papilloma, 1; pulmonary adenoma, 1; lymphoreticular sarcomas, 2; hepatic heman- gioma, 1; epidermal atrophy, focal hyperplasia, hyper- keratosis, acute and chron- ic inflammation; controls same as above	17
Mice (40)	"	- 40% Benzene	"	Pulmonary adenoma, 1; epi- dermal atrophy; focal hyp- erplasia, hyperkeratosis, acute and chronic inflamma- tion; controls same as above	17

TABLE XIII-5 (CONTINUED)

SUMMARY OF EFFECTS OF EXPOSURE TO ASPHALT ON ANIMALS

Species and Number	Route	Exposure		Effects	Ref- erence
		Amount, Strength, and Diluent	Frequency and Duration		
Mice (37)	Dermal	- 40% Benzene	1/wk 19 mon	Pulmonary adenoma, 1; epi- dermal atrophy, focal hyper- plasia, hyperkeratosis, acute and chronic inflamma- tion; controls same as above	17
Mice (50)	Subcuta- neous	200 mg - -	1*	Lung adenoma, 1; asphalt deposits covered by thin, relatively acellular sheath; no controls	32
"	"	200 mg - -	1**	Rhabdosarcomas, 2, 1 meta- static to lung and liver; skin accessory-structure tumor, 1 metastatic to lung; no controls	32
Mice (62)	"	0.2 ml 1% Olive oil	2/wk 41 wks, 1/wk until death	Rhabdosarcomas 1; fibrosar- comas, 7; no metastasis; 60 controls had no tumors	31
Mice (200)	im	0.1 ml 50% Tricapry- lin	Every 2 wk for 12 weeks	Sarcomas, 3; no tumors in 144 controls injected subcutaneously	19
Rats (120)	"	0.2 ml 50% Tricapry- lin	Every 2 wk for 24 wk	Sarcomas, 13; no controls	19

*After 111 d, 13 mice reinjected with 200 mg

**After 4 mon, 18 mice reinjected

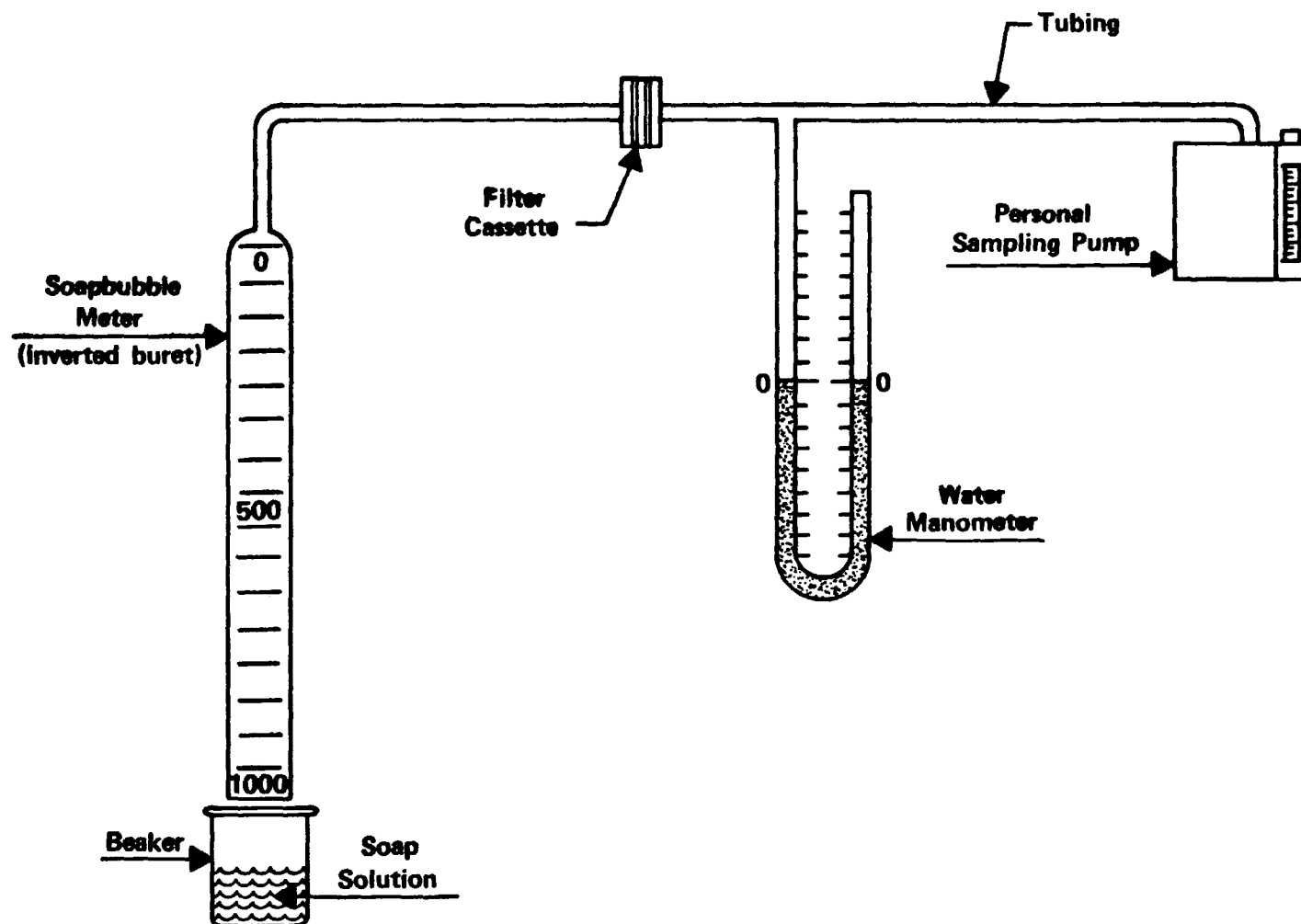


FIGURE XIII-2
CALIBRATION SETUP FOR PERSONAL SAMPLING WITH FILTER CASSETTE

